PURE NEURITIC HANSEN IN PATIENTS PRESENTING WITH PERIPHERAL NEUROPATHY-A CLINICOHISTOPATHOLOGICAL STUDY

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CERTIFICATE

Certified that this dissertation entitled ***"PURE NEURITIC HANSEN IN PATIENTS PRESENTING WITH PERIPHERAL NEUROPATHY-A CLINICOHISTOPATHOLOGICAL STUDY"*** is a bonafide work done by **DR. R. RAJESH,** Post Graduate Student of the Department of Dermatology, Venereology and Leprosy, Madras Medical College, Chennai – 600 003, during the academic year 2008 – 2011. This work has not previously formed the basis for the award of any degree.

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1. INTRODUCTION

Leprosy is one of the oldest diseases; endemic in India and of social concern as its complications leads to social stigma and decreased quality of life. Its long incubation period makes it completely difficult to diagnose early and its chronic course decreases the compliance of the patient towards the treatment. Leprosy is the most common treatable cause of neuropathy. The schwann cells of nerves are the first to be involved in leprosy. Leprosy is considered important primarily because of its potential to cause permanent and progressive physical deformities with serious social and economic consequences[1]. In all forms of leprosy atleast one peripheral nerve is invaded by M. leprae, though this may not be clinically evident. One of the primary complications of Leprosy is damage to peripheral nerves, which may be irreversible if not diagnosed and treated early. Leprosy, however can involve nerves without any skin changes[2]. Pure Neuritic Leprosy is a well accepted clinical entity. In the absence of skin lesions there is a greater possibility of missing the diagnosis of leprosy particularly due to a wide variety of pure neural manifestations that may mimic other peripheral neuropathies[6]. Classification of pure neuritic leprosy poses problems since the histological spectrum (e.g. Ridley-Jopling scale) is based chiefly on the skin findings. Review of the past literature shows conflicting reports about comparability of the skin and nerve findings[3]. Further the Pure Neuritic type is thought to be more common in countries endemic for Leprosy, particularly India. Hence this study tries to find out the prevalence and describes purely neuritic leprosy and its characteristics and the need for diagnosing it early.

2. REVIEW OF LITERATURE

Leprosy (Hansen's disease; Hanseniasis) is a chronic disease caused by Mycobacterium leprae, infectious in some cases and affecting the peripheral nervous system, the skin and certain other tissues[4]. Pure Neural Leprosy (Neural Leprosy) is a form of leprosy with one or more enlarged nerves, but no skin lesions[5].

PNL is defined as thickened peripheral nerve trunk with sensory loss in the area of its distribution, with or without associated motor paralysis in the absence of any skin patch regardless of clinical evidence of reaction involving nerve(s) [7].

2.1 Epidemiology:

Of the 254,525 new Leprosy cases detected globally during the year 2007-08; 137,685 (54%) were from India[8]. In India a total of 1.34 lakh new cases were detected during the year 2008-09, which gives Annual New Case Detection Rate (ANCDR) of 11.19 per 100,000 population. 32 States/ UTs have achieved the level of elimination i.e. PR less than 1 case per 10,000 population of which Tamilnadu is also one of the state[9].

The prevalence rate of leprosy for March 2009 in Tamilnadu with population of 67577617 (estimated as on Mar 2009) are 0.51[10].

District wise: Chennai has the highest prevalence rate of 0.73 and new cases detected being 546 during 2008-09 with percentage of new cases detected 0.92 in Chennai as per march 2009[11].

Pure Neuritic Leprosy is more common in places endemic for leprosy. Very few studies have been conducted for prevalence and incidence of Pure Neuritic Leprosy. One of the study done in rural areas of South India by Noordeen (1972) found the prevalence rate to be 8.2/1000 and it formed 18% of new cases in that area[12].

2.2 Historical Aspects:

Sushrata Samhita in 600BC classified Leprosy into three forms- Vat Rakta or Vat Shonita- disease with only sensory changes without any skin lesions, (Vat=Nerve Force, Rakta/Shonita= Blood) - Pure Neuritic . The forms with skin lesions were described under two varieties of Aruna Kushta; in one prominent symptoms were Sensory changes (non Lepromatous cutaneous lesions), and in the other ulceration (Lepromatous cutaneous lesion without much sensory changes)[13]. Wade, in 1952 was the first to recognize Polyneuritic cases as a seperate group[14, 15]. The Indian Classification of leprosy (1955) and its modified version have recognized it as a seperate group[14, 16]. Danielssen and Boeck (1848) were the first to record peripheral nerve involvement in leprosy. Neisser et al (1886) postulated that main trunk of the nerve was first affected and that infection spread centrifugally[17].

Khanolkar et al (1951) concluded that the cutaneous nerves are the earliest to be involved in all forms of Leprosy, he also put forward the hypothesis of M. leprae 'swimming upstream' via the axoplasm[18]. Dastur et al demonstrated that the free nerve endings were damaged more severely than the deeper perifascicular nerve fibres. The sensory nerve fibres were the first to be affected and sweating was the earliest function to be lost in leprosy[19]. Dastur et al further strengthened the idea of disto-proximal spread of infection along neural pathway[20]. Lumsden (1964) regarded the schwann cell as the most important cell involved in leprosy[21], this was further strengthened by studies done by Dastur et al (1973) [22].

2.3 Classification:

Pre-Manila Classification:

Danielssen and Boeck (1848) divided leprosy into Nodular and Anesthetic type[23]

Hansen and Looft (1895) divided leprosy into Tuberosa (nodular) and Maculoanesthetic[24]

Neisser (1903) divided into three forms: lepra tuberosa, lepra cutanae and lepra nervorum.

Manila Classification: In 1931 divided Leprosy into three types; Cutaneous, Neural, and Mixed.

Cairo Classification in 1938 divided leprosy into Lepromatous, Neural; Neural was further subdivided into Neuromacular Simple, Neuromacular Tuberculoid and Neuroanesthetic.

The Pan American Classification adopted classification on histological grounds and divided into Lepromatous, Tuberculoid (in place of neural) and Uncharacteristic type. Neural cases were split into the above types based on the histological findings.

The International Leprosy Congress in 1953 in Madrid included Pure neuritic type in both Indeterminate and Lepromatous type.

Indian Classification divided into six clinical forms:

Lepromatous (L), Tuberculoid (T), Maculoanesthetic (MA), Borderline(B), Polyneuritic (P) and Indeterminate (I).

In 1981, the Indian Association of Leprologists adopted Revised Indian Classification removing the Maculoanesthetic type and placing it as a variety of Tuberculoid type.

Job and Chacko further divided borderline group into Borderline Tuberculoid (BT) and Borderline Lepromatous (BL), retaining other types as in Revised Indian Classification.

Ridley and Jopling (1966) based on clinical, bacteriological, histological and immunological findings classified leprosy into five types[25]: Tuberculoid (TT), Borderline Tuberculoid (BT), Mid Borderline (BB), Borderline Lepromatous (BL) and Lepromatous Leprosy (LL). They further added new terms Indefinite Leproma(LI) and Indefinite Tuberculoid (TI).

WHO Classification: In 1982 for the purpose of treatment, WHO divided leprosy into two types primarily based on bacteriological findings: with few bacilli (<2+Paucibacillary) and relatively more bacilli (>/=2+ Multibacillary)[26]. In 1988, bacterial index was changed to zero for Paucibacillary and >/=1+ for Multibacillary. From then all smear positive cases were classified under Multibacillary and all smear negative cases as Paucibacillary [27].

2.4 Pathology:

Pathogenesis:

The exact mode of entry of M.leprae is not known. There are several possible ways M.leprae can infect a nerve.

1. M.leprae may enter through the naked axons in the epidermis or in the papillary layer of the superficial dermis. It then may travel up the axoplasm centripetally[28]

2. The organisms may be phagocytosed by the perineural cells of the nerves in the demis and from there invade the endoneurium and schwann cells.

3. The bacilli may be engulfed by schwann cells in the papillary dermis and then be transmitted from one schwann cell to another by contiguity.

4. M.leprae may enter the nerve through endoneural blood vessels during bacteremia[29, 30,31]

There are several reasons for selective destruction of nerves at the specified sites;

1. M.leprae multiply best at a temperature lower than core body Temperature [32]. M.leprae is prone to aggregate and grow inside nerve tissue at superficial sites where the temperature is relatively low.

2. Superficially placed nerves are easily traumatized by external injuries. The nerve damage following trauma may help to localize the infection at these areas.

3. These sites are mostly entrapment points in fibro-osseous canals causing the already inflamed and swollen nerves to the nerves, thus adding to the trauma sustained by them.

4. Swelling of the nerve due to edema and cellular infiltration under a tight unyielding perineurium produces an increase in

intraneural pressure, causing ischemia to nerve tissue. Demyelination and axonal degeneration are direct results of ischemia.

5. Vascular factors

- Histologic studies have identified vasculitic changes in small-sized arteries and arterioles (intraneural blood vessels) [33].
- Angiographic studies have revealed abnormalities in medium-sized vessels as well [34].
- The consequent ischemia may be one of the factors leading to neuropathy.

6. Genetic and immunologic factors

- The basis for the conspicuous destruction of nerve structure is thought to be a delayed hypersensitivity reaction with specific helper T cells reacting with M leprae [35].
- Leprae antigens are presented in the endoneurium by macrophages.
- Activation of macrophages leads to release of secretory products including neural proteases, potent oxidizing agents, and free radicals.
- A delayed hypersensitivity reaction in the endoneurium can cause major damage or even necrosis and intraneural abscesses.

7. Cytokines and chemical factors

- Interferon-gamma and interleukins
- Tumor necrosis factor (TNF) has been shown to induce demyelination; therefore, chronic production of TNF in lesions of leprosy may be related to some aspects of nerve damage

Pathology of Tuberculoid Nerves:

The Epitheloid Cell Granloma is the basis of the pathology of tuberculoid leprosy both in skin and in the Peripheral nerve. M.leprae lodges within the Schwannn cells at certain sites of predilection, viz. cooler temperature sites [38, 39]. When bacterial antigens are exposed and recognized by the host's immune cells, a granulomatous response develops as a result of the migration of lymphocytes and monocytes into the nerve. Tuberculoid leprosy due to the very localizing nature of the disease process starts as a longitudinal lesion involving one or few Fascicles of the nerve. Caseous necrosis can occur as a part of reactional or post reactional cases and is due to delayed hypersensitivity reaction to Mycobacterial antigens. Ridley (1988) concluded that the number and distribution of lymphocytes in a granuloma is a good index of the host's resistance to infection. Nerves with dense lymphocytic infiltrate indicates good CMI and collar of lymphocytes surrounding the well differentiated epithelioid cells and langhans type of giant cell is hallmark of established high resistant polar Tuberculoid lesions.

In severe involvement, multiple, well circumscribed epitheloid cell granulomas are seen within a highly enlarged fascicle, with a collar of lymphocytes with almost complete absence of neural elements. Myelinated fibres proximity to tubercles shows axonal degeneration. Granuloma consists of epitheloid cells, lymphocytes and langhans giant cells. The Perineurium is thickened with lymphocytic infiltrates. A few AFB are found in caseous material and in Schwann cells. Nerve abscess can occur, and consists of caseous necrosis surrounded by tuberculoid granuloma and fibrous tissue.

Pathology of Borderline Nerve Lesions:

Relatively heavy infiltration of both lymphocytes and macrophages, predominantly in the endoneurium, forming a dense collar at the Sub-Perineural region are seen. Most of the macrophages are bacillated. Dense aggregates of lympohcytes seen around some of the blood vessels suggests recent activity or impending reaction. There is total loss of myelinated fibres. Schwann cells are surrounded by a collar of inflammatory cells. AFB can be seen in macrophages and Schwann cells.

Pathology of Lepromatous Nerves:

Unlimited mulitplication of bacteria as a result of specific lack of CMI response of host is hallmark of polar Lepromatous Leprosy. The general architecture of nerve is usually preserved. Bacterial multiplication is seen

primarily in resident schwann cells. Absence of lymphocytic infiltrates is characteristic, but when present remain perivascular in early lesions. Epitheloid cells are absent, foamy macrophages can be found. Perineural fibrosis is seen in chronic cases with extensive fibre loss. Enlarged sub-perineurial zone are present in most fascicles. AFB are seen more often in this spectrum of nerves, more commonly in macrophages, schwann cells and endothellial cells.

Pathology of Pure Neuritic Lesions:

The entire spectrum of Leprosy including Indeterminate Leprosy have been found in the histopathological study of Pure Neural Leprosy [36]. Subpolar or Polar Lepromatous Leprosy types of lesions have not been recorded so far in PNL. Jacob and Mathai concluded that there is no interrelation between the nerve histopathology and clinical paramaters like number, distribution of affected nerves or the immune response. Major brunt of infection with M.leprae was borne by the non myelinated fibre schwann cells, therefore the loss of non-myelinated axons tends to be proportionately greater than that of the myelinated fibres. Many of the large fibres also showed paranodal demyelination and Remyelination.[37] AFB can be positive depending on the spectrum involved.

Perineural changes:

Thickening of perineurial sheath is caused by increased collagen and often oedema between perineural lamellae, with lymphocytic infiltration in Tuberculoid type.

Thickening of perineurium is due to increased numbers of lamellae of perineural cells in Lepromatous Leprosy. Perineurium initially infiltrated by lymphocytes leads to fibrosis in late stages with loss of perineural cells. Nerve damage is patchy with axonal degeneration and demyelination.

Blood vessel changes:

Active tuberculoid nerve lesions shows increased vascularity, obliteration of lumen due to swollen endothelial cells bulging into the lumen and marked reduplication of the endothelial basement membrane. AFB is usually absent

Lepromatous leprosy nerve lesions shows occlusion and obliteration of lumen. AFB are seen in the endothelial cells. Foamy macrophages may be seen perivascular.

Unusual features of leprosy neuropathy:

Fibrous long spacing collagen are seen in BT, BB, BL nerve lesions along with other proteinaceous granular matrix around the inflammatory cells.

Poly axonal Myelination- Myelination around a complex group of axons and schwann cell processes may be a result of defective schwann cell axon interaction resulting in abnormal myelination of parent unmyelinated fibre complexes [40, 41].

Renaut corpuscles may be seen in leprous nerves and Pure Neuritic lesions.

2.5 Clinical Features:

Incubation period:

The minimum incubation period for leprosy reported is as short as few weeks [42] to as long as 30 years or over, and the period shortens in endemic area. In south India the mean incubation period is 4.4 yrs [43]

Types of Nerve Involvement: Superficial peripheral nerves are most commonly involved in Leprosy. It can present as

Patch Anaesthesia: due to localised damage to cutaneous nerve as that occurs in Tuberculoid or Borderline Tuberculoid skin lesions.

Nerve Anaesthesia: due to damage to the peripheral nerve trunk, like that produced in the ulnar area of forearm and hand with or without skin lesions.

Diffuse Peripheral Anaesthesia: more common in Lepromatous Leprosy. Long standing damage to dermal nerves as well as to nerve trunks producing widespread peripheral anaesthesia more marked over the cooler parts of the body.

The last two types can occur in Pure Neuritic Leprosy.

Pattern of involvement:

Mononeuritis Multiplex involves two or more nerves in more than one extremity, e.g., left ulnar neuropathy and right peroneal neuropathy. This is seen in Leprosy, Diabetes mellitus and Vasculitic neuropathy.

A **Polyneuropathy** is a symmetrical, distal, usually ascending neuropathy due to involvement of the distal branches of nerves. Tingling, numbness, and pain, as well as sensory loss, occur in a symmetrical stocking or glove distribution in the feet or hands (Stocking-glove dysesthesia). Foot drop is common due to weakness of the lower leg muscles. More common in Lepromatous Leprosy.

Mononeuropathy is involvement of single nerve**.** The most common cause of Mononeuropathy is entrapment neuropathy due to the compression of a nerve in an anatomically narrow area. Pure Neuritic Leprosy may also present as Mononeuropathy. [60]

Size of Fiber:

Small-fiber neuropathy is a painful sensory neuropathy that occurs in Diabetes, Alcoholism, Amyloidosis, Leprosy, and AIDS.

Large-fiber neuropathy is characterized by motor weakness and loss of vibration and position sense. Most neuropathies are large-fiber neuropathies and are thus easily detectable by the nerve conduction study, which usually tests the large fibers of nerves. Leprosy is less common.

Clinical presentations of Pure Neuritic Leprosy

Leprosy can involve nerves without any skin changes [44]. Males are predominantly affected [1]. It is commonly seen in age group between 15 and 35yrs [45].Though less common can also occur in children [46]. Pure neuritic leprosy can be considered as early stage of leprosy as many patients with pure neuritic leprosy may develop skin lesions later, though spontaneous resolution is seen in patients with good CMI [48, 49]. Primary neuritic leprosy usually presents with signs and symptoms of nerve deficit which includes gradual motor weakness, sudden foot drop, or may present as anasthesia in an extremity or extremities discovered only when unrecognised injury leads to ulceration, a burn or cellulitis and osteomyelitis. Usually several nerves are involved.

The three physiological functions of nerve- Sensory, Motor and Autonomic may be affected. Sensory component is the earliest to involve and the most severely affected [50]. Rarely there is motor impairment with no sensory loss. Though Autonomic involvement is less marked compared to other functions, there can be associated loss of sweating and vasomotor dysfunction. Majority patients are mononeuritic [47].

Symptoms of leprous neuropathy usually include the following:

- Sensory symptoms such as diminution to complete loss of sensation, paresthesias in the distribution of affected nerves, and neuralgic pain when the nerve is struck or stretched

- Spontaneous blisters and Trophic ulcers consequent to sensory loss.

- Deformities due to weakness and wasting of muscles innervated by the affected peripheral nerves (e.g., claw hand or foot drop secondary to muscle weakness).

Sometimes painful nerves, and joints, tender lymphadenopathy can be presenting symptoms of reaction in nerves. A painless burn in an anaesthetic hand or foot may also be the first presenting symptom. Mononeuropathy and Mononeuritis multiplex can occur with the ulnar and common peroneal nerves most often involved [45,51]. Symmetric peripheral neuropathy also may occur. The commonly affected sensory nerves are the radial cutaneous, greater

Auricular and Sural nerves. The major mixed nerves that are most commonly affected include the ulnar, Median, Peroneal and facial nerve. Autonomic disturbances in form of loss of sweating and trophic changes can occur.

Other unusual presentations of Pure Neuritic Hansen:

- Nerve Abscess [52]
- Arthritis- Neuropathic joints and post-traumatic septic Arthritis are two most common presentations [53, 54].
- Tenosynovitis in combination with thickened nerves with or without symmetric Polyarthritis [55].
- Can also affect long bones as osteoporotic lesions and new bone formation, long before the development of skin lesions [56].
- Can also present with cranial nerve involvement [57].
- Generalised pruritus may occur very early in the course of the disease.
- Leprous Ganglionitis with loss of proprioception and vibration sensation can occur very rarely [58, 59].

2.6 Differential Diagnosis:

Mixed sensorimotor polyneuropathy suggests Nutritional neuropathy (due to Alcoholism, Beriberi, Vitamin B deficiency, or Pernicious anemia), Metabolic neuropathy (caused by Diabetes mellitus or Uremia), and Toxic neuropathy.

Sensory Polyneuropathy suggests a benign idiopathic sensory neuropathy, neuropathy related to diabetes or pernicious anemia, chronic sensory demyelinating neuropathy, and arsenic neuropathy.

Mononeuropathies similar to leprosy can also be caused by traumatic compression of individual nerves, Diabetes, Polyarteritis nodosa, Porphyria, Alcohol, Metal intoxication, Lupus Erythematosus.

Polyneuropathies can be produced by AIDS, Diabetic neuropathy, Systemic Amyloidosis, Alcoholic polyneuropathy, Lead intoxication, Arsenic[75].

	Common Causes of Peripheral Neuropathy:[74]
I	**Infections**
	i. Bacterial Leprosy Diphtheria Brucellosis
	ii. Viral Rabies Tropical Spastic Paraplegia (TSP) AIDS
	iii. Parasitic
II	**Malnutrition** i. Vitamin B deficiency Beri Beri Pellagra Vitamin B12 deficiency ii. Protein Calorie Malnutrition iii. Tropical Ataxic Neuropathy (TAN)
III	**Toxic** i. Chemical Organophosphate Induced Delayed Neuropathy Arsenical Neuropathy ii. Biological Bee and Wasp Stings Tick Bites
IV	**Genetically determined Neuropathies** Type I Familial Amyloid Polyneuropathy Hereditary Motor Sensory and Autonomic Neuropathies Acute Intermittent Porphyria Syringomyelia Acute Idiopathic Polyneuritis (Gullian Barre's Syndrome)
V	**Miscellaneous** Diabetes Mellitus Alcohol intoxication Polyarteritis nodosa Lupus Erythematosus

Conditions mimicking Pure Neuritic Hansen[82]:

Palpable nerve thickening without Anaesthesia or any nerve function impairment:

Excessive Muscular Development: Professional Wrestler, heavy weight lifters.

Pachydermoperiostosis

Palpable nerve thickening with Regional Anaesthesia, with or without muscle wasting

Primary Amyloidosis of Peripheral Nerve

Familial Hypertrophic Interstitial Neuritis (Djerine-Sottas Neuropathy)

Regional Anaesthesia with or without muscle wasting but with Palpable nerve thickening in some cases

Recurrent or Chronic Progressive (endotoxic) Polyneuritis-Acquired disorder of unknown cause.

Peroneal Muscular Atrophy (Charcot-Marie-Tooth type)

Regional Anaesthesia with or without muscle wasting but without palpable Nerve thickening

Syringomyelia

Tabes

Peripheral Neuropathy

Hereditary Sensory Radicular Neuropathy

Congenital Indifference to Pain

Hysteria

Trophic Ulcers

Diabetes Mellitus

Syringomyelia

Moorvan's Syndrome

Tabes Dosralis

Diastometamyelia

Thevanaurd Syndrome

Hereditary Sensory Neuropathy

Systemic Amyloidosis

Diabetic Neuropathy presents with paresthesias and pain in the lower limbs. Ankle reflex is absent. It is seen in chronic untreated patients. In more advanced stages, glove and stocking anaesthesia, impairment of sweating and vasomotor control and alterations of skin trophism with ulcerations can occur.

AIDS may present with slowly worsening peripheral neuropathy with painful paresthesias and weakness or paralysis of the distal muscles of extremities may occur. Hyporeflexia is also present.

Sarcoidosis may cause asymmetric polyneuropathy with hypertrophic infiltration and compression of cranial nerves and rarely radial and ulnar nerves. Other manifestations like hilar adenopathy, fever and uveitis may be present.

In **Systemic Amyloidosis** peripheral nerves can be affected in Primary and Amyloidosis with Myeloma. Symptoms are more commonly due compression of major nerve trunks in fibro-osseus channels. Impairment of autonomic function precedes the involvement of sensory fibres with loss of pain and temperature sensation. Histology shows features of amyloidosis.

Alcoholic Polyneuropathy involves sensory nerves with parasthesia and severe pain as early symptoms, later followed by glove and stocking anaesthesia. Other signs of chronic Alcoholism are present.in advanced stages. Areflexic distal parasthesia can occur in lower limbs.

Lead Polyneuropathy presents with slow progressive motor deficit with muscle atrophies and very minimal sensory loss. It is seen in patients with chronic lead exposure. Miners and typographers by occupation are more prone.

Early symptoms of **Arsenical Polyneuropathy** include parasthesias and cramps in lower limbs, followed by motor and sensory deficit, muscle atrophy. Deep reflexes are lost. Deformities of limbs can also occur. Seen in patients with chronic exposure to arsenic.

Drugs like Isoniazid, Nitrofurantoin, Disulfiram, Chloramphenicol, Metronidazole, Vincristine B, Dapsone, Stillbamidine, Thalidomide, Amiodarone may cause **Drug Induced Sensorimotor Polyneuropathy**.

Hereditary Sensory motor and autonomic Neuropathy:

Hereditary Sensory Neuropathy (Thevenard Syndrome, Familial acro-osteolysis), is a rare familial disorder. It is more common in childhood or early adult life. HMSN is associated with analgesia of lower limbs with neuropathic sequelae and nerve deafness. Pure osteolytic process without any evidence at bone regeneration which gradually extends proximally and eventually involves other acral bones. Shortening of foot is characteristic [76].

Five types of HMSN are described.

Type I is Autosomal dominant, seen in childhood and adolescent, with lower limb involvement. Involvement of peroneal muscles can lead to abnormal positioning of foot, high steppage gait and ulcers. Tendon reflexes are absent. Distal sensory loss is variable. Conduction velocity is reduced in affected nerves.

Type III (Djerine Sotta's syndrome) is Autosomal recessive, characterised by segmental demyleination of peripheral nerves. Nerves may be hypertrophied. Early symptoms occur in childhood with parasthesia and pain as main manifestation evolving into progressive symmetric weakness and deformities of lower limbs. Claw hand, glove and stocking anaesthesia may be seen. Tendon reflex is reduced or absent. Histopathology shows lack of cellular infiltration.

Acute intermittent Porphyria is an Autosomal dominant disorder presenting with asymmetrical polyneuropathy. Motor nerves with muscle atrophies are more common with minimal or no sensory loss. Other manifestations of porphyria are also seen.

Syringomyelia is a rare chronic degenerative disorder characterised by progressive development of cavities within central spinal cord. It is more common in the age group of 30-50. It may present with muscle wasting and segmental sensory loss of dissociated type. Upper limbs are more commonly affected.

Peroneal Muscular Atrophy (Charcot-Marie-Tooth type) is an inherited disorder in childhood with lower limb muscle weakness, hammer toes, pes cavus and callosities of feet. There is diminished or absent tendon reflexes.

In **Tabes Dorsalis** VDRL is reactive. There is dysfunction of posterior nerve roots with loss of sensation and position sense and difficulty in walking. Argyll-Robinson's pupil is seen and Romberg's sign is positive.

Acute Idiopathic Polyneuritis (Gullian Barre's Syndrome) affects peripheral and cranial nerves, with thickening of nerve trunks. Histologically nerve is affected throughout with inflammatory cells. Onset of disease follows viral illness, vaccinations or serotherapy. Tendon reflexes are absent. CSF albumin concentration is raised.

Congenital indifference to pain is a sensory disorder with intact or normal cutaneous and nervous system [77]. Patients with congenital indifference to pain may present with repeated trauma, fractures or self mutilation. Normal defensive response to pain sensation is defective or absent but the response to other sensations is normal. Skin or nervous system are histologically normal[78] and improves with age.

Pachydermoperiostosis is a condition where there is generalised thickening of skin, periosteum and bone along with generalised thickening of nerves. Clubbing of fingers are also seen.

2.7 Complications:

One of the major complications of Pure Neuritic Leprosy is development of deformities.

Type of leprosy, duration of disease and number of nerves involved influence the development of deformities.

Deformities can be of three types; specific, paralytic and Anaesthetic type

Paralytic and anaesthetic type is seen in pure neuritic leprosy.

Paralytic Deformities:

- Face:
    - Lagophthalmos and Facial Palsy
- Hand:
    - Claw hand
    - Wrist Drop
    - Paralytic Thumb
- Foot:
    - Foot Drop
    - Claw Toes
    - Inversion of Foot

Claw hand is the most frequent deformity in leprosy and is due to ulnar paralysis (partial/Ulnar Claw hand) or due to both Median and Ulnar paralysis (Total/Complete Claw hand). Nerve trunk paralysis in upper limb follows a spatiotemporal pattern. Ulnar nerve may be affected alone or may precede paralysis of other upper limb nerves. Whereas median nerve paralysis occur in association with ulnar nerve. Radial nerve paralysis is almost always preceded by ulnar and median nerve paralysis. Rarely triple paralysis can occur involving ulnar, median and Radial nerve.

Ulnar nerve damage leads to paralysis and atrophy of all interosseous muscles, lumbricals of ring and little fingers and all hypothenar muscles.

Ulnar claw hand is characterised by:

- Hyper extension at the Metacarpal phalangeal joints and flexion at Interphalangeal joints of fourth and fifth finger.
- Flattening of hypothenar eminence
- Wasting of interossei, clinically seen as depression on the dorsa of the hand
- Flattening of thenar eminence on the distal part (flexor pollicis brevis)
- Straightening of thumb at metacarpophalangeal joint (flexor pollicis brevis).
- Flattening of medial aspect of forearm (Flexor carpi ulnaris muscle paralysis)

Median nerve is very rarely damaged in isolation. There is paralysis and atrophy of lumbricals of middle and index finger along with thenar muscle atrophy.

In Paralytic Thumb, combined ulnar and median nerve damage paralyzes all small muscles of thumb, and the thumb is activated solely by the extrinsic muscles. The thumb collapses in zigzag fashion. In addition to flexor pollicis brevis, adductor pollicis is also paralyzed.

Patients with radial nerve paralysis, already have median and ulnar paralysis (Triple paralysis). There is inability to dorsiflex the wrist.

The paralysis of Common Peroneal nerve leads to paralysis of muscles of foot (Tibialis anterior, Extensor hallucis, extensor digitorum and the peroneal muscles). Patient is unable to evert and dorsiflex the foot leading to foot drop and high stepping Gait.

Paralysis of Intrinsic muscles of foot give rise to Claw foot.it is not a serious deformity, but it indicates planar intrinsic muscle paralysis and hence increased risk of ulceration under metatarsal heads and tips of the toes.

Paralytic deformities of Face are rare and facial nerve is the most common cranial nerve damaged in leprosy. Isolated cranial nerve involvement is very rare, though multiple cranial involvement may occur very rarely (Polyneuritis Cranialis) [79]. The common deformity of face includes

Lagophthalmos and Facial Nerve palsy, individual branch of Facial nerve may be affected.Later Exposure keratitis and corneal ulcer can occur in a patient with lagophthalmos.

Anesthetic Deformities:

Sensory loss and paralysis can lead to profound changes in the equilibrium conditions of articular system of the extremities and expose unsual surface to pressure and wear and tear. This can lead to anesthetic deformities.

- Hand:
    - Ulceration
    - Blisters, erosion
    - Softening and mutilation of digits
    - Contractures
- Foot:
    - Trophic Ulcers
    - Tissue scars
    - Callosity
    - Cellulitis and edema

Trophic ulcer over the plantar aspect is the most common Anesthetic deformity. Plantar ulcer most often occurs under the proximal phalanx of great toe and the first metatarsal head. The heel and mid lateral border of sole can also be affected. Collapse of arch of foot can lead to ulceration of central part of the sole.

Trophic ulcer can result from three reasons:

- Walking on insensitive feet
- Infection following penetrating injury
- Infection through a deep fissure in dry insensitive sole or through a minute crack associated with corn or callosity

Trophic ulcer can be:

- Acute- inflamed
- chronic
- complicated- with osteomyelitis, deep infections, gas gangrene
- Reccurrent

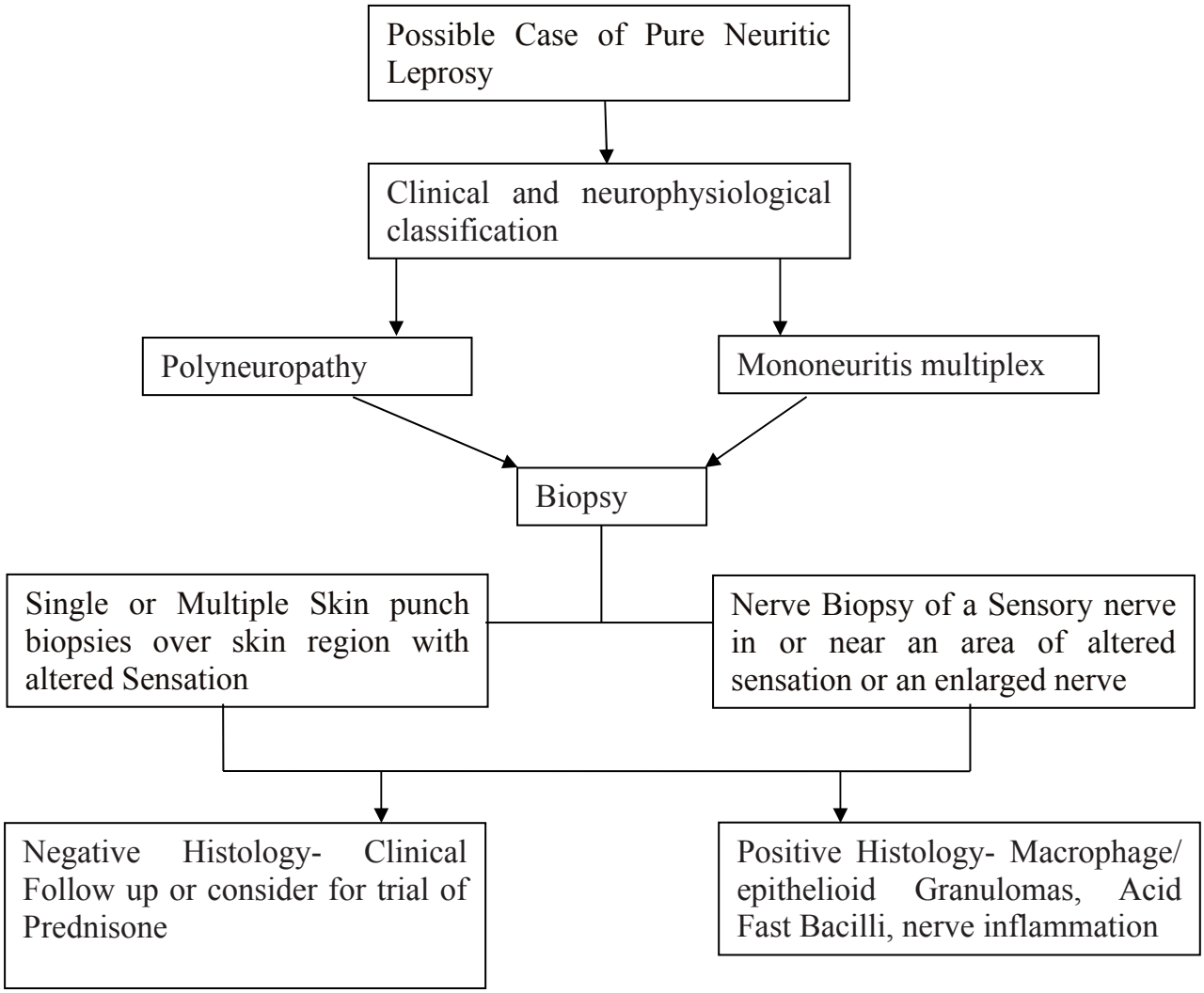
Malignant transformation particularly Squamous Cell Carcinoma have been reported in longstanding Trophic Ulcers [80, 81].

WHO Disability Grading [27]:

Hands and Feet	
Grade 0	No Anesthesia. No visible Deformity or Damage
Grade 1	Anesthesia present but no visible deformity or damage
Grade 2	Visible deformity or damage present
Eyes	
Grade 0	No eye problem due to leprosy, no evidence of visual loss
Grade 1	Eye problem due to leprosy present. But vision not severely affected (Vision 6/60, can count fingers at 6 meters)
Grade 2	Severe visual impairment (vision worse than 6/60, inability to count fingers at 6 meters. Also includes lagophthalmos, iridiocyclitis and corneal opacities)

2.8 INVESTIGATIONS:

Approach to a Case of Pure Neuritic Hansen [70]:



Slit Skin Smear:

Sites: both Ear lobes.

The chosen area is cleaned with a small cotton spirit swab. Once the spirit dries, the skin is pinched up into a fold between the index finger and thumb. Enough pressure should be applied to stop or minimise bleeding. With help of sterile scalpel (no.11) a 5mm long and about 2mm deep cut is made. The blunt edge of the blade is used to scrape the side of the cut. If blood exudes, it should be wiped off with a dry small cotton swab. The material collected is smeared over a clean Glass slide. The slide is then subjected for Ziehl-Neelsen Stain for AFB. The slide is heat fixed and covered with freshly filtered dilute Carbol Fuschin and allowed to stand for 15 minutes. It is then washed gently under tap water. Acid ethanol (Acid Alcohol) mixture is then run over the slide by keeping the slide at an angle, till the fluid becomes pink to colourless. The slide is again washed gently under the tap water and counter stain (Methylene Blue) is covered over the smear alone for 1 minute and washed gently. The smear is then kept to dry. The slide is examined under Oil immersion.

Atleast 100 microscopic fields should be examined before reading is done. **Bateriological Index** [61]**:**

Average number of AFB	**Bacteriological Index (BI)**
0/100 fields	0
1-10/100 fields	1+
1-10/10 fields	2+
1-10/in an average field	3+
10-100/in an average field	4+
100-1000/in an average field	5+
Many clumps of bacilli in an average field (>1000)	6+

Nerve Conduction Study (NCS)

The nerve conduction study (NCS) is the most essential part of the work-up in patients with a peripheral neuropathy [71, 72]. This study helps confirm peripheral neuropathy, determine the type of neuropathy, localize the site of lesion or entrapment, and follow the course of the disease. The nerve conduction study includes motor and sensory nerve conduction tests. NCS is a technique in which electrodes are placed over nerves, either sensory, motor or mixed nerves and stimulated with a stimulator. NCS can be done separately for motor nerves, sensory nerves and mixed nerves. Sensory nerve conduction is a more sensitive index than motor nerve conduction in the diagnosis of peripheral neuropathy. Nerve conduction is abnormal in peripheral neuropathy, but it is normal in myopathy and anterior horn cell disease. The nerve conduction study is useful in early detection of nerve damage in leprosy even in clinically normal nerves of leprosy patients [73]. NCS identifies the neuropathy in 76 to 80% of patients with diabetic neuropathy [71]. It is important to remember that the NCS could be normal in a few patients with mild neuropathy of axonal degeneration especially in small-fiber neuropathy. The hallmark of nerve conduction abnormalities in axonal degeneration is a diminution of the amplitude of the Compound muscle action potential (CMAP) and compound nerve action potential (CNAP) in the presence of normal or near-normal maximal nerve conduction velocity (NCV). On the other hand, the hallmark of nerve conduction abnormalities in demyelinating neuropathy are conduction block, abnormal temporal dispersion (dispersion phenomenon), and marked slowing in the Nerve conduction velocity.

Mechanism

When a motor nerve is stimulated, the impulses are conducted across the nerve and reach the muscle. 2 electrodes are placed over the concerned muscle for eg abductor pollicis brevis for median nerve, one active electrode over the muscle belly and reference electrode over its tendon and the nerve is stimulated at 2 sites, one distally and another proximally. In case of median nerve, electrode is placed at the wrist and elbow respectively. The distance between the 2 sites are measured and can be used for analysis. The various parameters that can be assessed are latency, amplitude of the wave, duration of the wave & nerve conduction velocity. Similarly for sensory nerve, such as median nerve, stimulus is given at the wrist and recording electrodes are placed over the index finger. Commonly ring electrodes are used for sensory conduction studies involving fingers since they are better conductors. This method of doing sensory NCS is called anti-dromic studies as it is recorded against the normal direction of impulse propagation.

General NCS

General NCS involves motor and sensory nerve conduction of median, ulnar and radial nerves in upper limb and post tibial and common peroneal nerve motor conduction studies and sural sensory studies of lower limb. Waves obtained in motor NCS are called Compound Muscle Action Potentials [CMAP] and in sensory nerves are called Sensory Nerve Action Potentials [SNAP]

Interpretation

Prolonged distal latencies, conduction blocks, conduction slowing, decreased nerve conduction velocities, prolonged duration and temporal dispersion of the waves are hallmarks of a **demyelinating pathology**. Significantly reduced amplitudes without prolonged distal latencies are typical of **axonal pathology**. However both can overlap depending on the nature and duration of illness. Leprosy initially starts as a Demyelinating , followed by consequent Axonal degeneration.

NERVE BIOPSY

TYPES OF NERVE BIOPSY:

There are two types of nerve biopsy: **Fascicular biopsy** and **Whole biopsy**.

In fascicular biopsy, only a few fascicles of the nerve are biopsied in order to lessen permanent sensory loss and long-term dysesthesia [63]. Follow up for 5 years didn't not reveal any difference in sensory loss between fascicular and whole nerve biopsy [64].

There is more chance of missing Tuberculoid Type in Fascicular biopsy, as it affects only few fascicles. So most Centres prefer Whole Nerve biopsy.

SURAL NERVE BIOPSY

Biopsies of three different nerves have been described:

Radial Cutaneous nerve.

Superficial Peroneal nerve.

Sural nerve.

The sural nerve biopsy is preferable for four reasons:

(1) is easily identifiable and relatively protected from compression injury because it is located behind the lateral malleolus

(2) is purely sensory, thus producing no motor deficit following biopsy

(3) is liable to be affected by neuropathy because it is a distal branch of a long nerve

(4) is easily tested electrophysiologically.

PROCEDURE

In a sural nerve biopsy, the patient is placed in the lateral decubitus position and a pillow is placed under the ankle to be biopsied. The skin incision is made under local anesthesia with 1% lidocaine behind the lateral malleolus and halfway between the posterior aspect of the Achilles tendon and the lateral malleolus. This skin incision is extended proximally for 4 to 5 cm, parallel to the Achilles tendon. Under the incised skin, the lesser saphenous veins are usually seen. The whitish pearly sural nerve is identified medially under the lesser saphenous veins. When the sural nerve is touched by an instrument, the patient often feels a shooting electrical pain — a definite sign that the structure is the sural nerve. Both the nerve and the veins are superficial to the deep fascia. Once the sural nerve is identified, the nerve is anesthetized prior to cutting with a small amount of lidocaine a few millimeters proximal to the in-

tended transection site in order to prevent pain when the nerve is cut. Once total anesthesia is achieved, the nerve is firmly clamped proximal to the transection site. This most likely reduces the likelihood of any potential post-biopsy neuralgia. Nevertheless, the patient should be warned that there may be a possible sharp pain at the moment the nerve is cut.

Generally, the degree of pain is inversely proportional to the severity of the neuropathy[65]. The proximal nerve is lifted gently and cut with sharp dissection distal to the hard clamping. A nerve segment at least 4 cm in length should be obtained with due care in order to avoid any unnecessary trauma to the nerve. The superficial fascia and skin incision is closed using interrupted mattress skin sutures with 4-0 coated vicryl sutures inside and 3-0 nylon sutures outside. An elastic bandage is applied locally to reduce the accumulation of blood and fluid. Sitting with the leg in a dependent position for long periods, excessive walking, or running are discouraged. Sutures may be removed in 7 to 10 days. A mild Analgesic and an antibiotic are prescribed for postoperative care.

SEQUELAE OF NERVE BIOPSY

Following the sural nerve biopsy, there is invariably a sensory loss over the lateral aspect of the foot corresponding to the sural nerve territory. This area gradually decreases in size, but a quarter-sized area remains permanently insensitive to pin-prick. Some immediate postoperative pain is not uncommon

[66]. However, pain gradually diminishes over time [67]. Serious reactions following the sural nerve biopsy are rare. Significant pain or paresthesia are noted in 10% of patients 1 year after the biopsy [66]. Asbury and Connolly noted serious side-effects in only 2 of 103 patients: post-traumatic neuroma in 1 and pain in the other [68].

Sample Processing:

The nerve is cut into 4 sections with a sharp razor, to be processed for paraffin, frozen, semithin, and electron microscopic (EM) sections, and for fiber teasing. The piece closest to the transection site should be used for paraffin sections, and the midportions utilized for semithin, EM sections and frozen sections. This distribution is preferred because potential cutting artifacts are not that critical for paraffin sections or nerve fiber teasing, whereas artifact-free sections are essential for semithin and EM sections.

For paraffin sections, the nerve is processed by fixing it in a neutral buffered formalin solution. It is then cut into two pieces, one-third for the transverse section and two-thirds for the longitudinal section. Sections are cut at 5 μm, except for Congo-red stain, which should be cut at 8 to 10 μm, and are then stained with H and E, modified Fite Faraco stain.

Modified Fite Faraco stain [69]:

Both Nerve and skin tissue can be stained using modified fite faraco for Acid Fast Bacilli.

1. Warm sections and de-paraffinize in a mixture of two parts xylene/one part vegetable oil for 15 mins.
2. Blot dry and wash in water. Repeat if any xylene-oil remain on the section.
3. Filter on carbol fuchsin solution, DO NOT HEAT, for 20 mins.
4. Wash in running tap water.
5. Differentiate in 10.0% sulphuric acid for 2 mins.
6. Wash well in running tap water, rinse distilled water.
7. Counterstain in 0.25% methylene blue for 20 seconds. it is important not to overstain .
8. Wash and blot dry. Do not dehydrate in alcohol.
9. Clear in xylene. Repeat the blotting-xylene treatment until section is clear.
10. Mount in a DPX type mountant.

SKIN BIOPSY:

Skin biopsy is not much useful in Pure Neuritic Leprosy. Even though many studies have demonstrated few histopathological changes in skin [62], the findings were non-specific inflammation in the dermis and peineural inflammation in few. Most of these biopsies were done on area of sensory loss[45].

Jacob and Mathai found negative skin biopsy in PNL and half of the patients were confirmed with nerve biopsy [36].

3. AIM OF THE STUDY

1. To find the Age group distribution of Pure Neuritic Leprosy.

2. Gender characteristic patterns of Pure Neurtic Hansen.

3. Geographical distribution of Pure Neuritic Hansen

4. Occupational incidence among patients with Pure Neuritic Hansen

5. Clinical presentations of Pure Neuritic Hansen.

6. Pattern of nerve involvement.

7. Deformities associated with Pure Neuritic Hansen.

8. Electrophysiological Pattern of Pure Neuritic Hansen.

9. Histopathological changes in the Nerve associated with Pure Neuritic Hansen.

4. MATERIALS AND METHODS

4.1 Study Design:

A cross-sectional Study was conducted during the period of August 2008 to August 2010. Ethical Committee Approval was taken.

4.2 Study Sample:

Ninety patients presenting with peripheral neuropathy symptoms and suspected Pure Neuritic Leprosy, attending the Department of Dermatology and Leprosy, Government General Hospital, Chennai were selected for study.

4.3 Subjects:

Written consent was taken for the study.

Inclusion Criteria:

New onset Pure Neuritic Leprosy.

Suspected Cases either self reporting or referred from other departments.

Patients without any leprosy skin lesions.

All patients with sensory loss of an area or part of the body.

Patients with motor loss or motor weakness.

Patients with nerve thickening, Nerve tenderness, Nerve swelling or Abcess.

Patients with deformities like Claw hand, Foot drop, Wrist drop, Facial palsy, Lagophthalmos without any skin lesions or history of previous cutaneous Leprosy.

Patients with Trophic ulcer.

Released from Treatment with onset of new nerve symptoms

Exclusion Criteria:

Patients already on treatment.

Patients with skin lesions of leprosy.

Patients with previous history of leprosy skin lesions.

Patients with history of treatment for previous Leprosy skin lesions.

4.4 Clinical Profile:

For every patient, with the help of Pre-designed Proforma, Demographic profile (Age, Sex, Occupation, Residence) were noted, detailed history pertaining to the presenting symptoms, duration, history of treatment and history of household contact were taken, a thorough clinical examination was done to rule out the presence of any hypopigmented anaesthetic skin lesions, nodules or other signs of Cutaneous Leprosy and also to rule out other causes of Peripheral neuropathy. Number of nerves involved, Sensory or Motor deficit, Trophic ulcer and Deformities were examined. Disabiltiy Grading was done based on WHO Disability Grade (1988).

Skin Smears from both ear lobes were taken for Acid Fast bacilli demonstration. Routine investigations like Complete Blood Count, Random Blood Sugar, Liver Function Test, Renal Function Test, VDRL, HIV-ELISA, Chest X Ray, ECG were taken.

MRI LS Spine and MRI Brain were taken in selected patients as per Neurologist's advice.

4.5 Electrophysiological Study:

A written Consent was taken. Nerve Conduction Study was done to detect the electrophysiological properties of affected Nerves. Neurology opinion was taken for the Nerve Conduction Study in each patient.

4.6 Histopathological examination:

A written consent was taken. Left Sural Nerve Biopsy was done under aseptic precautions under local anaesthesia and Hematoxylin and Eosin stain and modified Fite Faraco Staining were done. The histopatological changes were studied with respect to Nerve tissue damage (Fibre/Axon loss or Degeneration, Perineural thickening and Fibrosis), Cellular component (Epitheloid Cells, Giant Cells and Foam Cells), Pattern of inflammation (Granuloma, Diffuse infiltrates, focal or Sparse infiltrates, Perivascular infiltrates and Necrosis) and Acid Fast Bacilli demonstration by Fite Faraco.

Skin Biopsy was done in selected patients with areas of sensory deficit. The biopsied specimens were subjected for Hematoxylin and Eosin stain and modified Fite Faraco Staining.

5. OBSERVATIONS

Ninety patients were selected for the study, of which only fifty patients gave consent for nerve biopsy. All the cases were followed up throughout the study period. Data collected were analysed. The following characteristics were studied.

1. Age Distribution
2. Gender Distribution
3. Occupation
4. Geographical Distribution
5. Clinical Presentation
6. Duration of Disease
7. Nerve Involvement
8. Deformities
9. Disability Grade
10. Electrophysiological Pattern
11. Histopathological Profile

I. Age and Sex Distribution:

Age	Sex		Total
	Male	Female	
<15	1	2	3
15-25yrs	11	4	15
26-35yrs	**19**	9	28
36-45yrs	17	4	21
46-55yrs	17	2	19
>55yrs	1	3	4
Total	66	24	90

Male predominated with 66 men (73.3%) presenting with Pure Neuritic Leprosy and most of the patients were in the age group of 26-35years (n=28, 31.1%). Male to Female ratio of 2.75:1 was observed (fig 1.).

II. Occupation Distribution:

The occupational classification was modified from Registrar General's Occupational Classification.

Occupation	Number of Patients (n=87)
Professional Group	1 (1.1%)
Non Manual Skilled Worker	5 (5.6%)
Manual Skilled Worker	29 (32.2%)
Semi-Skilled Worker	24 (26.7%)
Unskilled Worker	9 (10.0%)
Student	3 (3.3%)
Housewife	15 (16.7%)
Unemployed	1 (1.1%)
Total	87

Majority of the Patients were Manual Skilled worker, (n=29, 32.2%) followed by Semiskilled workers , (n=24, 26.7%). 3(3.3%) cases were children hence not included above (fig 2.).

III. Geographical Distribution:

Location	**Number of Patients(n=90)**
Chennai	29 (32.2%)
Kancheepuram	7 (7.8%)
Thiruvallur	28 (31.1%)
Thiruvanamalai	3 (3.3%)
Vellore	9 (10.0%)
Rest of TN	8 (8.9%)
Immigrants	6 (6.7%)
Total	90

29 patients (32.2%) were from Chennai followed by Thiruvallur district, 28 cases (31.1%). About 6 cases (6.7%) were immigrants to Chennai (fig 3.).

IV. Presenting Complaints:

Presenting Complaints	**Number of Patients (n=90)**
Sensory Complaints	**70 (77.8%)**
Hypoesthesia	54 (60%)
Parasthesia	10 (11.1%)
Hyperesthesia	6 (6.7%)
Motor Complaints	**44 (48.9%)**
Deformities	**29 (32.2%)**
Trophic Changes	**16 (17.8%)**

Sensory deficit, (n=70, 77.8%) was the most common presenting complaint of which Hypoesthesia was the most common complaint. (n=10, 11.1%) presented with parasthesia (fig 4.).

V. Duration:

Age	Duration (n=90)				
	<6mnths	6mnth-1yr	1-2yrs	2-5yrs	>5yrs
<15yrs	3	0	0	0	0
15-25yrs	5	5	3	1	1
26-35yrs	2	14	4	1	7
36-45yrs	5	9	2	2	3
46-55yrs	6	5	3	2	3
>55yrs	0	2	1	1	0
Total	21	35	13	7	14

Duration of Pure Neuritic Hansen in majority of patients were less than 1 year, 35 cases (38.9)

VI. Age of Onset of Pure Neuritic Hansen:

	Age of Onset	**Age at the time of presentation**
	Number of Patients	**Number of Patients**
<15 years	4 (4.4%)	3(3.3%)
15-25 years	24 (26.7%)	15(16.7%)
26-35 years	23 (25.6%)	28 (31.1%)
36-45 years	19 (21.1%)	21 (23.3%)
46-55 years	16 (17.8%)	19 (21.1%)
>55 years	4 (4.4%)	4 (4.4%)
Total	90	90

After reducing the duration of disease from the Age at the time of presentation, the actual Age of Onset of Disease was calculated. 24 (26.7%) Patients developed Pure Neuritic Hansen in the Age group of 15-25 years (fig 5.).

VII. Nerve Involvement:

Cutaneous Nerves	Unilateral	Bilateral	Total
Greater Auricular Nerve	7 (7.8%)	24 (26.7%)	31 (34.5%)
Radial Cutaneous Nerve	21 (23.3%)	46 (51.1%)	**67 (74.4%)**
Sural Nerve	14 (15.6%)	51 (56.7%)	65 (72.3%)
Truncal Nerves			
Ulnar Nerve	25 (27.8%)	61 (67.8%)	**86 (95.6%)**
Lateral Popliteal Nerve	7 (7.8%)	66 (73.3%)	73 (81.1%)
Posterior Tibial Nerve	14 (15.6%)	53 (58.9%)	67 (74.5%)

Radial Cutaneous Nerve (n=67, 74.4%) was the most common cutaneous nerve involved followed by Sural Nerve. Ulnar Nerve (n=86, 95.6%) was the most common Truncal Nerve involved followed by Lateral Popliteal Nerve. One patient presented with multiple cutaneous nerve enlargement (P13 a & b). Overlap is observed.

VIII. Pattern of Nerve Involvement:

i. Nerve Function involved:

Age	Functional deficit (n=90)			
	Nil	Sensory	Sensorimotor	Motor
<15yrs	0	3	0	0
15-25yrs	1	7	5	2
26-35yrs	0	16	12	0
36-45yrs	0	14	7	0
46-55yrs	0	7	10	2
>55yrs	0	2	2	0
Total	1	49	36	4

Most patients had Sensory deficit predominantly followed by sensorimotor loss. Only 4 patients had purely motor deficit.

ii. Clinical Pattern involved:

Age	Nil	Mononeuropathy	Mononeuritis Multiplex	Polyneuropathy
<15yrs	0	3	0	0
15-25yrs	1	14	0	0
26-35yrs	0	13	12	3
36-45yrs	0	16	4	1
46-55yrs	0	17	2	0
>55yrs	0	3	1	0
Total	1	66	19	4

Mononeuropathy was the most common clinical pattern of Nerve involvement, followed by Mononeuritis Multiplex. Four Cases had distal Polyneuropathy.

IX. Deformities:

i. Paralytic Deformities:

Paralytic Deformities	Unilateral	Bilateral	Total
Facial Nerve Palsy/Lagophthalmos	0	1(1.1%)	1
Claw Hand	12(13.3%)	7(7.8%)	19
Wrist Drop	1(1.1%)	1(1.1%)	2
Foot Drop	8(8.9%)	5(5.6%)	13
Claw Toes	0	1(1.1%)	1

19 patients had claw hand, of which 7 had bilateral . one patient with bilateral Lagophthalmos was also seen.

ii. Anaesthetic Deformities:

Anaesthetic Deformities	Number of Patients
Trophic Ulcer	22(24.4%)
Resorption	6 (6.7%)
Auto-amputation	1 (1.1%)
Spontaneous Blistering	1 (1.1%)

Trophic ulcer was the most common anaesthetic deformity observed, followed by Resorption.

X. Disabiltiy:

Age	Disability (n=90)		
	grade 0	grade 1	grade 2
<15yrs	0	1	2
15-25yrs	1	11	3
26-35yrs	0	12	16
36-45yrs	0	13	8
46-55yrs	0	5	14
>55yrs	0	1	3
Total	1	43	**46**

Most patients with Pure Neuritic Hansen had Grade 2 Disability with visible deformities.

XI. Nerve Conduction Study:

Nerve Conduction Study was conducted only in 50 patients as rest of the patients didn't give a written consent.

Nerve Conduction Study	Number of Patients (n=50)
Normal	12 (24.0%)
Sensory Neuropathy	14 (28.0%)
Motor Neuropathy	7 (14.0%)
Sensorimotor Neuropathy	17 (34.0%)
Total	50

Nerve Conduction study in Pure Neuritic Hansen showed Sensorimotor Neuropathy in majority of the Patients, 17(34%).

i. Relation to Duration of Hansen:

	Nerve Conduction studies (n=50)			
Duration	**Normal**	**Sensory Neuropathy**	**Motor Neuropathy**	**Sensorimotor Neuropathy**
<6mnths	5	3	2	2
6mnth-1yr	4	8	3	6
1-2yrs	2	1	0	4
2-5yrs	0	0	2	2
>5yrs	1	2	0	3
Total	12	14	7	17

NCS showed normal study in 5 patients with a duration less than 6 months followed by 4 patients with duration of disease within 6months-1 year. 7 patients out of 12 patients with duration of disease less than 6 months had NCS abnormality (fig 7.).

ii. Relation to Clinical Pattern of Nerve involvement:

Clinical Involvement	Nerve Conduction studies (n=50)				
	Normal	**Sensory Neuropathy**	**Motor Neuropathy**	**Sensorimotor Neuropathy**	**Total**
Mononeuropathy	**11**	10	7	12	40
Mononeuritis Multiplex	1	4	0	5	10
Polyneuropathy	0	0	0	0	0
Total	12	14	7	17	50

Of 40 patients with Mononeuropathy , NCS showed normal conduction in 11 Patients. Among 10 patients with Mononeuritis Multiplex , 4 had sensory conduction defects and 5 patients had sensorimotor conduction defects. One didn't show any conduction abnormality.

XII. Histopathological Study:

i. Nerve Histopathology:

Nerve Histopathology	**Number of Patients (n=50)**
Nerve Tissue Damage	
Axonal Loss /Degeneration	14 (28.0%)
Fibrosis	8 (16.0%)
Perineural Thickening	12 (24.0%)
Cellular profile	
Epitheloid Cells	13 (26.0%)
Giant Cells	2 (4.0%)
Foam cells	7 (14.0%)
Pattern of Iniltration	
Granuloma	5 (10.0%)
Diffuse Infiltration	7 (14.0%)
Focal/Sparse Infiltration	20 (40.0%)
Necrosis	1 (2.0%)
Perivascular inflammation	9 (18.0%)
AFB (Modified Fite Faraco)	3 (6.0%)

Among the 50 patients subjected for Nerve Biopsy (n=20, 40%) patients showed Focal or Sparse inflammatory infiltrates in the nerve tissue, (n= 13, 26%) patients showed epitheloid cells. Granuloma was seen in (n=5, 10%) patients and (n=14, 28%) patients had axonal degeneration. Overlap of findings observed.

ii. Histopathological Spectrum in Nerve:

Diagnosis	Number of patients (n=50)
Normal	3 (6.0%)
Non Specific	7 (14.0%)
Tuberculoid (TT)	26 (52.0%)
Borderline Tuberculoid (BT)	10 (20.0%)
Borderline Lepromatous (BL)	1 (2.0%)
Lepromatous Leprosy (LL)	3 (6.0%)
Total	50

26 (52%) patients were typed to Tuberculoid spectrum based on the histopathological findings in the nerve tissue, followed by Borderline Tuberculoid Spectrum, (n= 10, 20%) patients (fig 6.).

iii. Relation to Duration of Hansen:

Duration	Neuro-Histopathological Changes (n=50)					
	Normal	Non Specific	TT	BT	BL	LL
<6mnths	0	3	7	1	0	1
6mnth-1yr	3	2	9	6	0	1
1-2yrs	0	0	5	1	1	0
2-5yrs	0	1	2	1	0	0
>5yrs	0	1	3	1	0	1
Total	3	7	26	10	1	3

Only 3 patients with Pure Neuritic Hansen showed normal histological findings in the Nerve tissue. 7 patients had Non Specfic findings suggesting, but not specific for Pure Neuritic Hansen. All the 12 Patients with duration of disease less than 6 months showed histological changes (fig 7.).

iv. Relation to Clinical Pattern of Nerve Involvement:

Clinical Pattern of Nerve Involvement	Neuro-Histopathological Changes (n=50)					
	Normal	Non Specific	TT	BT	BL	LL
Mononeuropathy	3	5	23	7	1	1
Mononeuritis Multiplex	0	2	3	3	0	2
Polyneuropathy	0	0	0	0	0	0

3 patients with Mononeuropathy showed normal Nerve histology and 5 patients showed Non Specific changes. One patient had changes suggestive of Lepromatous Leprosy.

V. Skin Biopsy:

15 patients with areas of sensory loss were subjected to skin biopsy. Most of the patients showed normal histology with only 3 patients showing sparse inflammatory infiltrates along the dermal nerves. Fite faraco were negative in all the skin biopsy specimen.

vi. Slit Skin Smear:

Slit Skin Smear for AFB taken from both ear lobes in all patients were negative.

6. DISCUSSION

Though incidence of Leprosy is slowly decreasing, the need for Pure Neuritic Leprosy has become imperative, especially in endemic country like India. Very few studies have been reported till now in context to Pure Neuritic Hansen. In this study, Ninety patients clinically diagnosed to have Pure Neuritic Hansen were studied during the period of August 2008 to August 2010.

i. Age Distribution:

Kumar et al reported 65(4.2%) cases in a retrospective study of 1542 cases, of which majority belonged to the age group of 15-35 years.[84] The present study showed similar findings where, 28 (31.1%) patients belonged to 26-35 Age group followed by 21(23.3%) patients of Age group 36-45 at the time of presentation. The youngest patient in this study was a female child of age 5 years and the oldest being 64 years old female. Three patients with pure Neuritic Leprosy were children below 15 years.

Age of onset of Pure Neuritic Leprosy was 15-25years, 24 (26.7%) patients followed by the age group 26-35 years, 23 (25.6%). So an overall age group of 15-35 years was observed similar to the previous studies.

ii. Sex Distribution:

Kumar et al reported a male predominance with a male: female sex ratio of 2.6:1.[84] Male predominated in this study also, with 66 (73.3%) patients presenting with the disease. Male to female Ratio of 2.75:1 was observed. Only 24 (26.7%) females presented with PNL.

iii. Occupation Distribution:

Most of the patients with PNL were Manual skilled worker, 29 (32.2%) patients, followed by Semi Skilled Worker, 24 (26.7%) patients. Only one patient from Professional group presented with the disease. 15 (16.7%) housewives also had Pure Neuritic Leprosy .

The increased risk of trauma to peripheral nerves may be one of the reason for increased incidence in manual worker. The Occupational Classification was modified from Registrar General's Occupational Classification, in which skilled Manual workers included carpenters, Plumber, Electrician, etc., and Semi skilled workers included factory assembly workers, Farmers, etc.

iv. Geographical Distribution:

Highest number of patients reported to our OPD were from Chennai, 29 (32.2%) patients, followed by Thiruvallur, 28 (31.1%). Other places were

district surrounding Chennai, one of the reason for this being easy accessibility. 6 (6.7%) patients who had PNL were immigrants from other states of which Bihar predominated. Three immigrants had developed symptoms of disease long before coming to Chennai. Among Thiruvallur district, Patients from Thiruvallur and Gumdipoondi were more common.

v. Presenting Complaints:

Kumar et al observed multiple symptoms at the time of presentation, sensory deficit was most common, followed by motor symptoms and Trophic changes.[84] Similar results have been observed in this study with 70 (77.8%) patients presenting with Sensory complaints of which 49 (54.4%) patients had sensory impairment as the only Presenting Complaint.

54 (60%) patients had hypoesthesia and 10 (11.1%) patients had parasthesia. About 6 (6.7%) patients complained of pain along the course of nerves during their first visit. 44 (48.9%) patients had motor complaints such as weakness or difficulty in doing daily activities. 29 patients (32.2%) patients presented with Deformities as their first complaint. 16 (17.8%) patients presented with ulcer over extremities as their complaint.

vi. Duration:

In a study done in Delhi by Mendiratta et al, majority had a complaints for less than a year (56.2%) further supporting the present study.[85] In this

study, Majority of patients had duration of onset of Neuritic symptoms within one year, 56 (62.2%) patients, of which 21 (23.3%) patients had onset within 6 months.

vii. Nerve Involvement:

Among the Truncal Nerves, Ulnar Nerve was commonly thickened, followed by Lateral Popliteal in a study done by Kumar et al.[84] About 86 (95.6%) patients had thickening of Ulnar Nerve, of which 51(56.7%) showed bilateral uniform thickening. Three Patients had tenderness along the course of Ulnar Nerve and one Patient had nodular thickening. Of the three patients, one had only unlilateral uniform thickening and tenderness. Radial Cutaneous Nerve was the most common cutaneous nerve involved with 67 (74.4%) showing thickening of the nerve.

viii. Pattern of Nerve Involvement:

i. Nerve Function deficit:

49 (54.4%) patient had Sensory deficit alone, followed by 36 (40%) patients with Sensorimotor deficit.

ii. Clinical Pattern of Nerve Involvement:

Mononeuropathy was reported by Kumar et al as the most common presentation in his study.[84]

Mononeuropathy was the most common clinical manifestation in this study also, 66 (73.3%) patients followed by Mononeuritis Multiplex, 19 (21.1%) patients.

ix. Deformities:

i. Paralytic Deformities:

Claw hand was the predominant paralytic deformity with 12 (13.3%) patients presenting with unilateral and 7 (7.8%) patients with bilateral Claw Hand. 13(14.4%) patients had Foot drop of which 5 (5.6%) had bilateral involvement.

ii. **Anaesthetic Deformities**:

Trophic ulcer predominated the anaesthetic deformities, 22 (24.4%) patients. One patient had Auto-amputation of index finger.

x. Disability:

Majority of the patients with Pure Neuritic Hansen were classified into Grade 2 Disabitlity with visible deformities. Since Pure Neuritic Hansen affects predominantly the Peripheral Nerve, it is less likely to see a PNL patient with Grade 0 where there is no sensory deficit or visible deformity.

xi. Nerve Conduction Study:

Of ninety patients, only fifty patients gave a written consent for Nerve Conduction study as well as Biopsy. Conduction Studies Showed abnormality in 38 (76%) patients and it was normal in 12 (24%) patients. NCS was unable to detect any abnormality in Nine patients who had developed the disease recently (within one year). It also failed to show abnormalities in eleven patients with disease localised to one extremity (Mononeuropathy). One of the reason for this would be because, PNL, particularly Tuberculoid may affect one or few fascicles of nerve and may lead to Small fibre neuropathy in the early stage, which is difficult to diagnose.

xii. Histopathological Study:

i. Nerve Histopathology:

Jardim et al demonstrated inflammatory infiltrate composed of epithelioid granuloma (42.1%), mononuclear infiltrate (36.8%) or macrophages positive for bacilli (21%) and Fibrosis in 78.9% of the biopsies in nineteen patients with PNL. He also reported acid fast bacilli in 16%, and nonspecific inflammatory infiltrate and/or fibrosis in 39% in his previous study.[87, 88]

Of the fifty patients who were subjected to Nerve Biopsy, Tuberculoid spectrum was the most common histological type diagnosed, 26 (52%) cases, of which 16 (32%) had less than one year duration of disease. Three patients were diagnosed to have Lepromatous Leprosy with foam cells and Acid Fast Bacilli. Fibrosis was seen in 8 (16%) patients. 13 (26%) patients had epitheloid cell infiltration and only 5 (10%) patients had Granuloma. Three patients didn't show any histopathological abnormality (Mononeuropathy). All the 3 cases showed abnormality in Conduction study and had recent onset of disease (less than a year).

ii. Skin Histopathology:

Menicucci et al conducted skin biopsy in sensory altered skin of clinically diagnosed PNL, and demonstrated Borderline tuberculoid changes in 31% cases. 33% cases showed non specific skin changes.[86] Suneetha et al in her retrospective study of 182 cases, reported 29 cases to develop skin lesion later in life, of which 11 cases developed within one year of diagnosis of PNL. She further concluded that PNL may be a preceding stage of Cutaneous Leprosy.[83]

15 patients with areas of sensory loss were biopsied and the tissue sections showed sparse Perineural inflammatory cells in the dermis in 3 patients. Acid Fast bacilli could not be demonstrated.

Two patients, during the one year period of study, developed anaesthetic patches which were consistent with Hansen histopathology of Tuberculoid spectrum.

xiii. Relapse:

Haimanot et al demonstrated granulomatous infiltration and diffuse inflammatory infiltrates and some cases of Acid Fast Bacilli in Sural Nerve tissue sample in patients who were released from treatment for a long time suggesting a Relapse.[89]

In this study, eight patients had history of Anti Leprosy Treatment in the past for similar complaints. Six patients had MDT-PB Adult dosage, of which 4 patients had completed the treatment for 6 months, and 2 patient discontinued within first three months. Two patients had history of Dapsone therapy for one year. Histopathology of Nerve in five of these patients revealed dense infiltrate in one patient released from treatment, BT in another patient who completed treatment and TT and Non specific changes in the patient with incomplete MDT. The patient with history of Dapsone therapy in the past showed evidence of perivascular infiltrates and few foam cells. The other three were not subjected to biopsy and were assessed clinically.

Relapse though uncommon can occur, even in Pure Neuritic Hansen. Since PNL can present histopathologically as any spectrum of Leprosy, it becomes necessary to assess the patient histologically also prior starting the MDT, and the decision of considering PNL as Paucibacillary or Multibacillary is incomplete unless, it is supported by NCS and histopathlogical findings.

7. CONCLUSION

- The Commonest Age group of occurrence is 25-35 years, though actual Age of onset of PNL is 15-25 years.
- Males predominates Females with a Male to Female Sex Ratio of 2.75:1
- PNL presents with more than one symptom, and the most common manifestation of PNL is Sensory impairment, especially hypoesthesia.
- Ulnar Nerve is the most common nerve trunk involved in PNL and Radial Cutaneous Nerve is the most common cutaneous nerve to get thickened. Bilateral thickening is more common.
- Pure Sensory involvement is more common, both sensory and motor involvement can occur, but pure motor involvement is very rare.
- Mononeuropathy is most common in PNL, though PNL can present as Mononeuritis Multiplex, polyneuropathy is very rare.
- Claw Hand is the most common Paralytic Deformity. In lower limb, Foot drop is more common.
- Trophic Ulcers of foot are the most common Anaesthetic Deformity. Auto-amputation and Resorption can occur in PNL.
- PNL most commonly presents with Grade 2 disability and is a major health concern as it is associated with severe social dependence and morbidity.

- Nerve conduction is an important Screening tool for PNL, but has limited use in very early and localised disease.

- All histological types of Leprosy can be demonstrated in PNL, but Tuberculoid is the most common and Lepromatous Leprosy may occur very rarely.

- AFB may be negative in Nerve biopsy, especially in Tuberculoid spectrum.

- Skin Biopsy in PNL has limited use alone though it may be an adjunct for Nerve biopsy in cases with sensory altered skin.

- Nerve biopsy is the gold standard in diagnosis of PNL, though it may miss very early stage of PNL and further detailed histopathological studies are needed.

- Pure Neuritic Hansen can be an initial stage of Leprosy, which may manifest later in life.

- Though Relapse is uncommon, Multi Drug Therapy based on clinical signs alone may lead to inadequate therapy and increased risk of Relapse as the PNL can histopathologically present as any spectrum of Leprosy.

- In endemic country like India, further larger studies are needed for early diagnosis and treatment of PNL, and thus preventing serious deformities and morbidity.

Abbreviations for Master Chart

ALT	
H/O	Anti Leprosy Therapy History
B/L	Bilateral
BL	Borderline Lepromatous
BT	Borderline Tuberculoid
CH	Claw Hand
D/G.I	Diffuse/Gross Infiltration
E.C	Epitheloid Cells
F	Female
Fi	Fibrosis
F.C	Foam Cells
F/S.I	Focal/ Sparse Infiltration
FD	Wrist Drop
FP/L	Facial Palsy/Lagophthalmos
G.C	Giant Cells
GAN	Greater Auricular Nerve
Gr	Granuloma
LL	Lepromatous Leprosy
LPN	Lateral Popliteal Nerve
Lt	Left
M	Male
MMn	Mononeuritis Multiplex
Mn	Mononeuropathy
MN	Motor Neuropathy
N	No
NCS	Nerve Conduction Study
ndlr	nodular
NFL/D	Nerve Fibre Loss/Degeneration
NS	Non Specific
Occ	Occasional
OHA	Oral Hypoglycemic Control
Po	Polyneuropathy
PT	Perineural thickening
PTN	Posterior Tibial Nerve
PVI	Perivascular Infiltration
RCN	Radial Cutaneous Nerve
Resorp tn	Resorption
Rt	Right

S	Sensory Neuropathy
SAN	Sensory Axonal Neuropathy
SM	Sensorimotor Neropathy
SMAN	Sensory motor Axonal Neuropathy
SN	Sural Nerve
SSS-AFB	Slit skin Smear for Acid Fast Bacilli
T	tender
Tr.Ulcer	Trophic Ulcer
TT	Tuberculoid
U/L	Unilateral
UN	Ulnar Nerve
Y	Yes

CHARTS

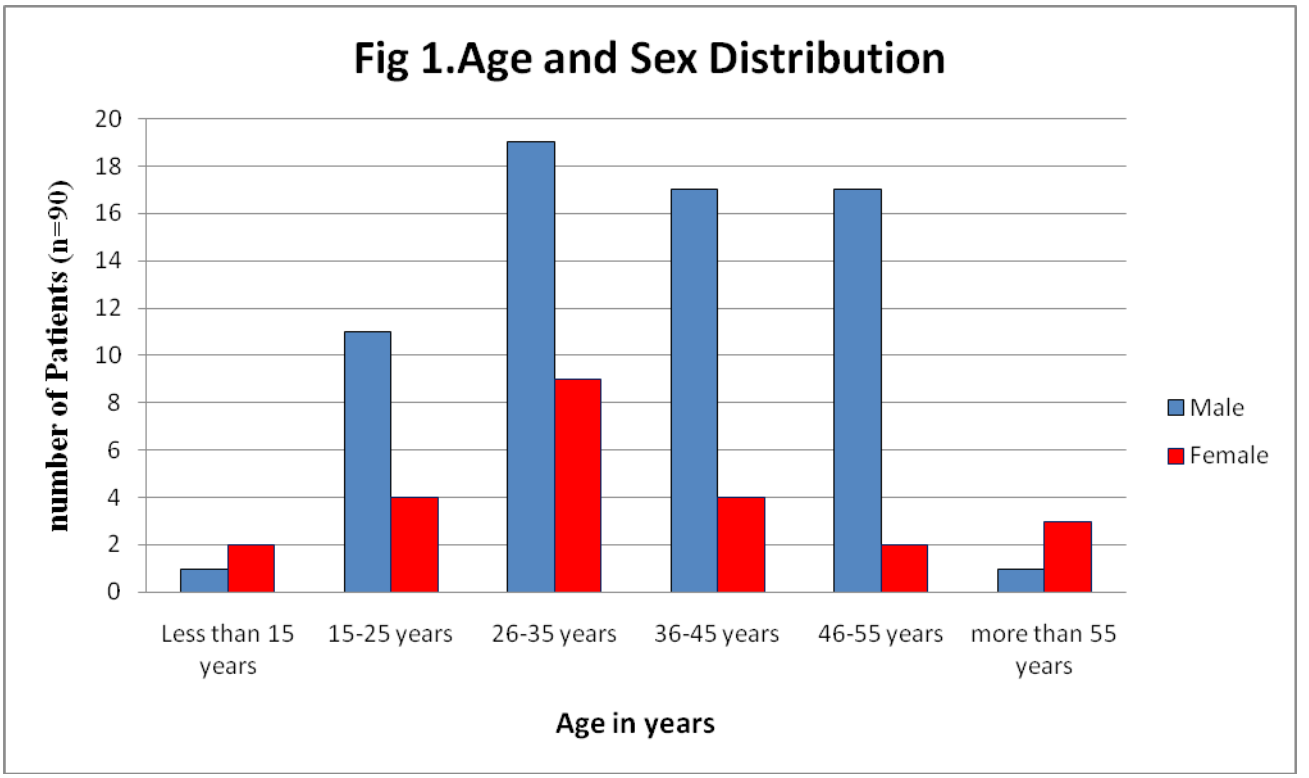


Fig 1.Age and Sex Distribution
number of Patients (n=90)
20
18
16
14
12
10
8
6
4
2
0
Less than 15 years
15-25 years
26-35 years
36-45 years
46-55 years
more than 55 years
Age in years
Male
Female

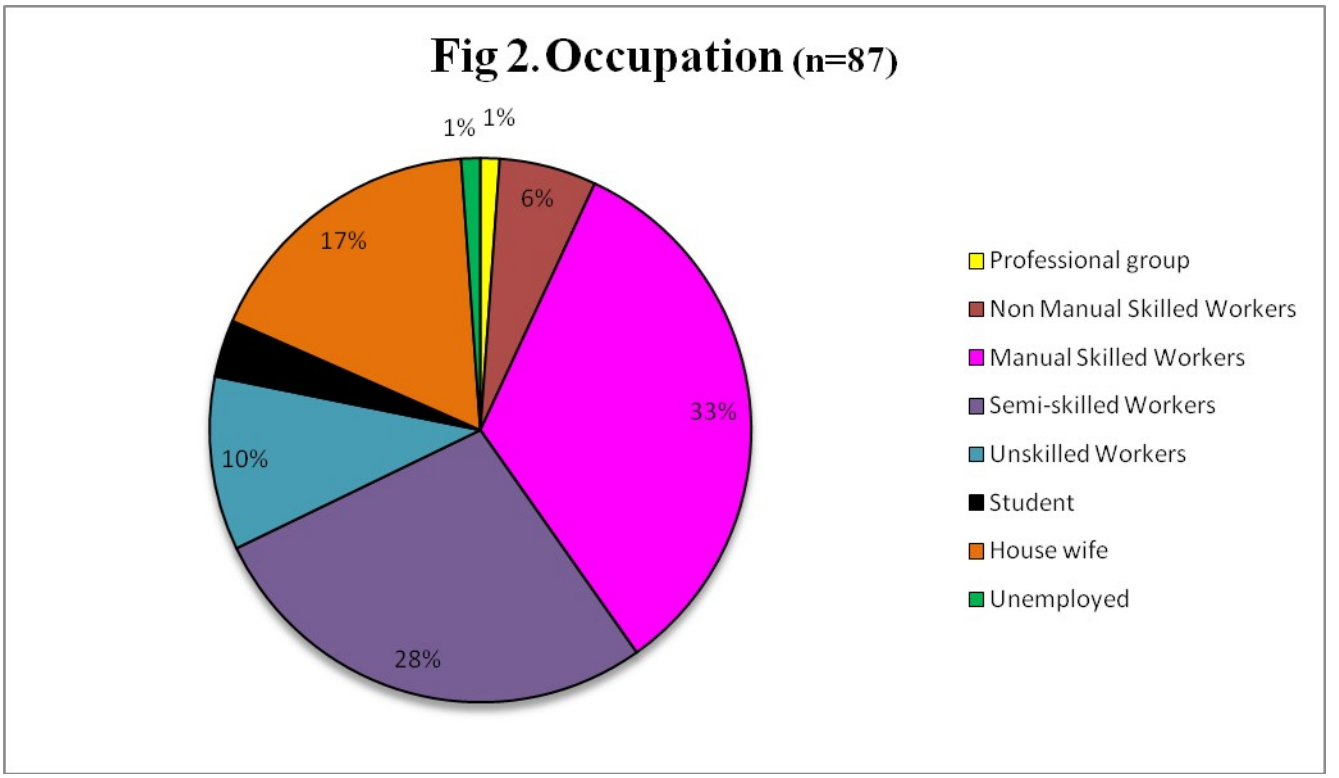


Fig 2. Occupation (n=87)
1%
1%
6%
17%
33%
10%
28%
Professional group
Non Manual Skilled Workers
Manual Skilled Workers
Semi-skilled Workers
Unskilled Workers
Student
House wife
Unemployed

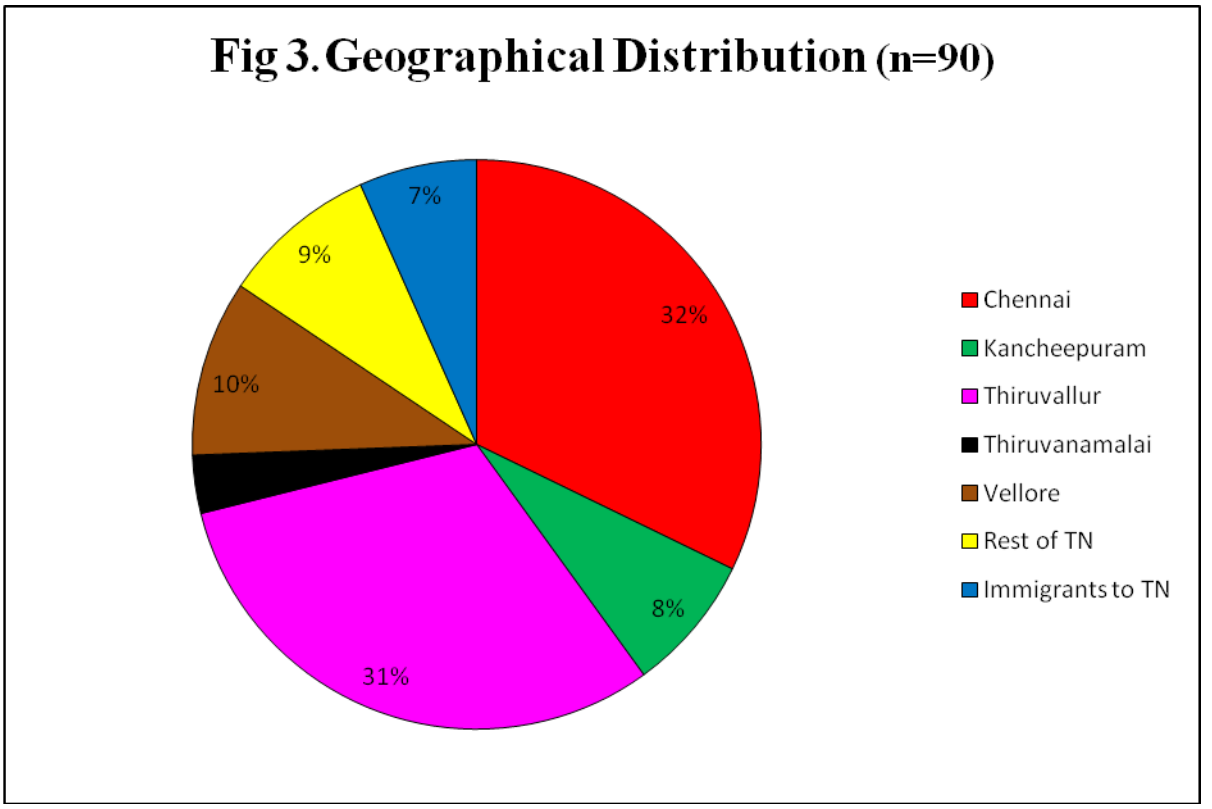
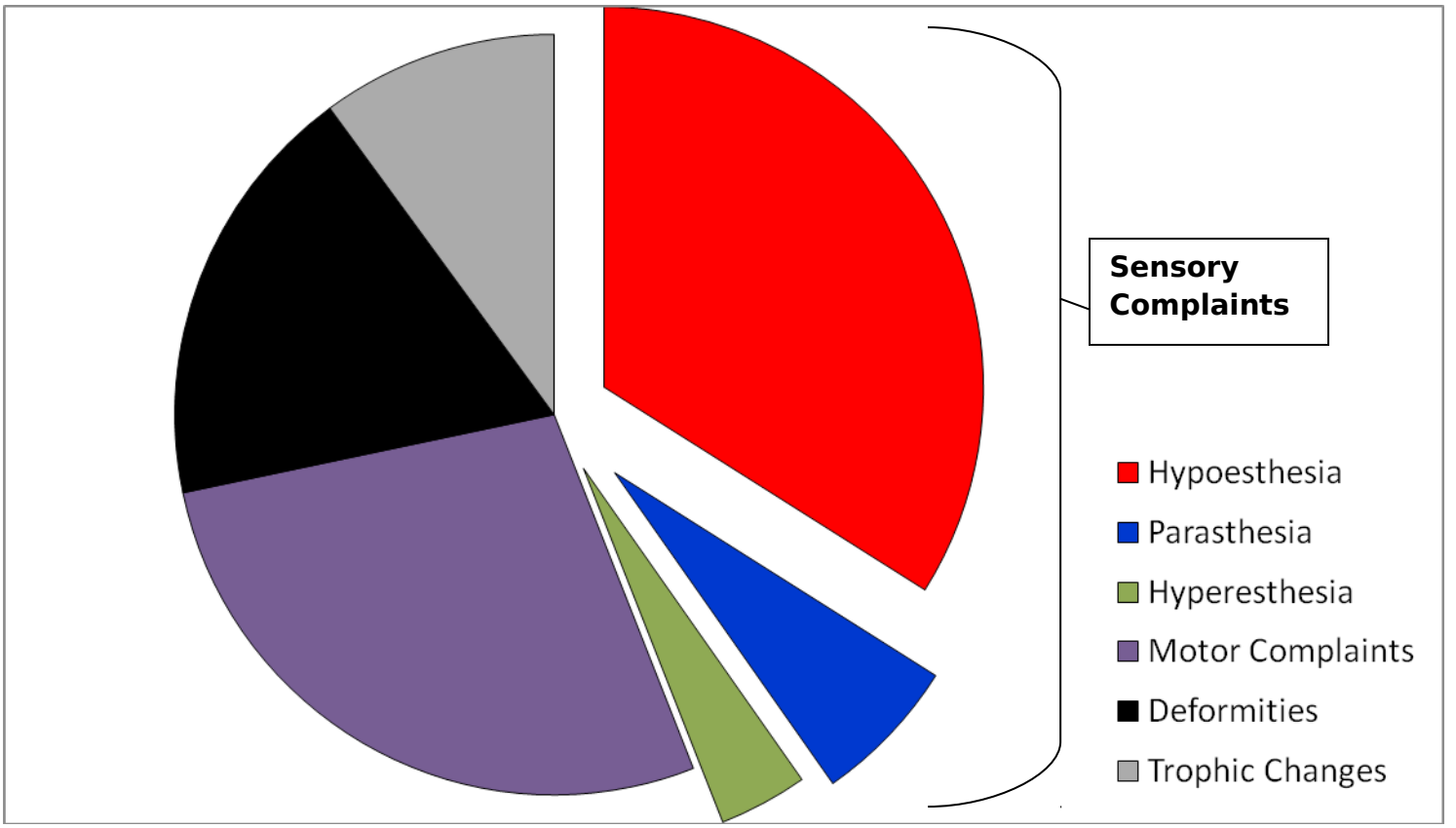


Fig 4.Presenting Complaints



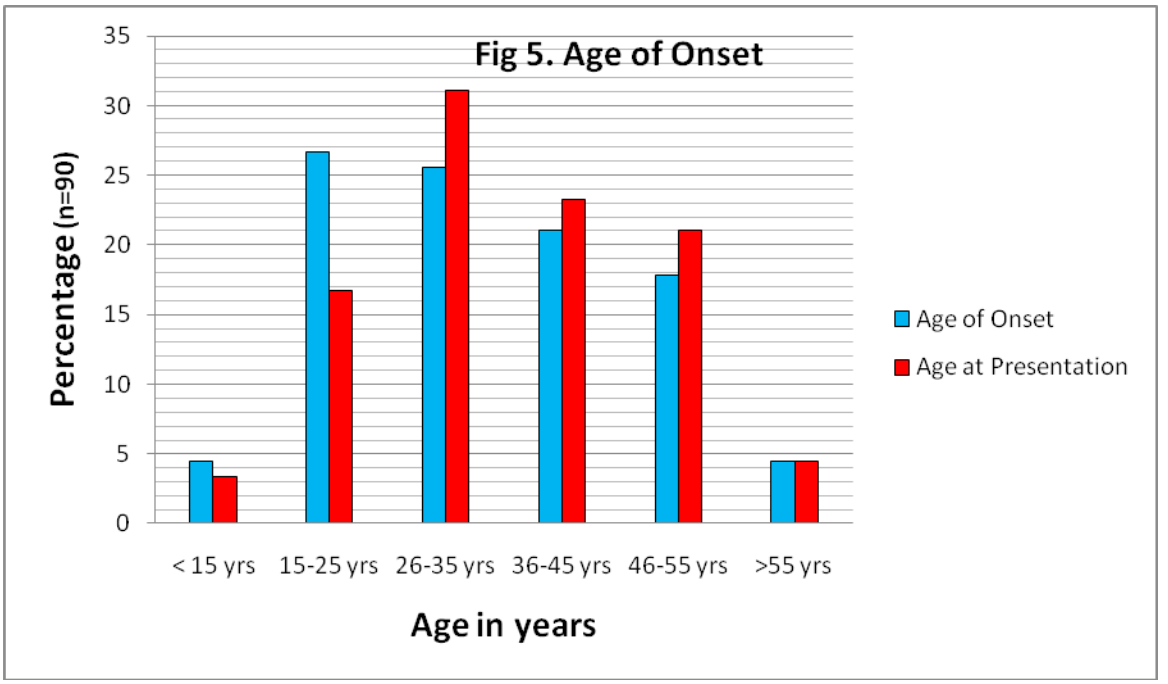


Fig 5. Age of Onset
Percentage (n=90)
35
30
25
20
15
10
5
0
< 15 yrs
15-25 yrs
26-35 yrs
36-45 yrs
46-55 yrs
>55 yrs
Age in years
Age of Onset
Age at Presentation

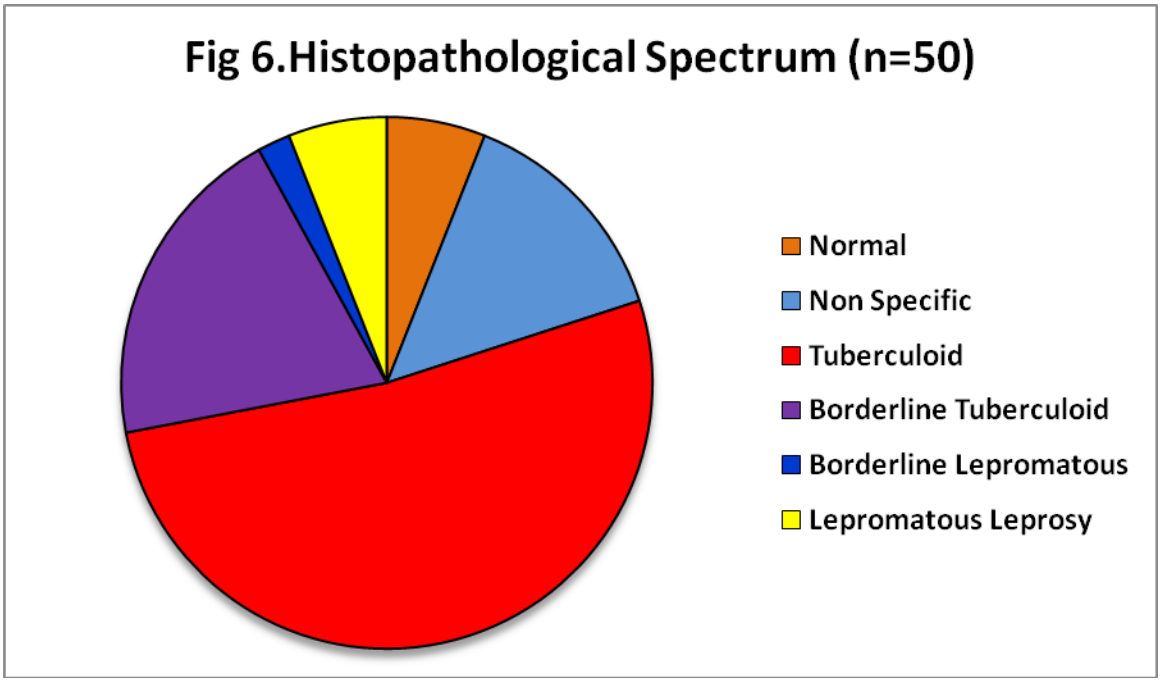


Fig 6.Histopathological Spectrum (n=50)
Normal
Non Specific
Tuberculoid
Borderline Tuberculoid
Borderline Lepromatous
Lepromatous Leprosy

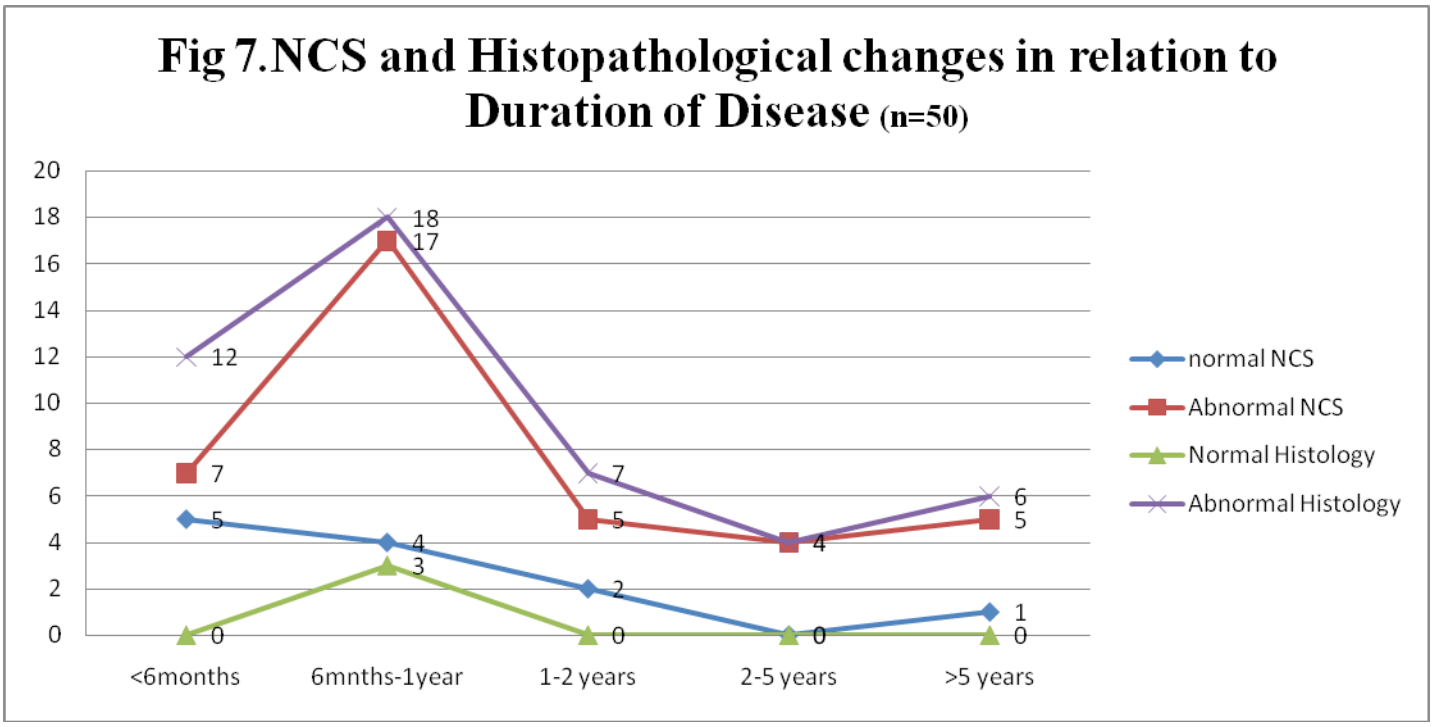


Fig 7.NCS and Histopathological changes in relation to Duration of Disease (n=50)
20
18
16
14
12
10
8
6
4
2
0
18
17
12
7
7
5
5
6
5
4
4
3
2
1
0
0
0
0
<6months
6mnths-1year
1-2 years
2-5 years
>5 years
normal NCS
Abnormal NCS
Normal Histology
Abnormal Histology

sl.no	Cases	Age	Sex	Occupation	Address	presenting complaints					Cutn. Ns involved			Nerve Trunks involved			type of nerve involvement			Deformities						Disability	comorbidity			ALT H/O
		(yrs)				Sensory Complaints	extremity involved	Motor Complaints	Deformities	duration	GAN	RCN	SN	UN	LPN	PTN	N. Funct	Clinical Pattern	Wasting	CH	FD	WD	FP/L	Tr.Ulcer	Resorptn	(WHO 1988)	DM	Alcohol	smoking	
1	Case 1	36	M	farmer	villipuram	nil	both limbs	nil	Y	6yrs	nil	B/L	B/L	B/L, tndr	B/L	B/L	SM	MMn	N	U/L	U/L	N	B/L	Y	N	Grade 2	NIL	Occ	Occ	
2	Case 2	13	M	student	thiruvanamalai	hypoasthesia	upper limb	nil	N	4months	nil	nil	Rt>Lt	Rt>LT	nil	B/L	S	Mn	N	N	N	N	N	N	N	grade 1	NIL	NIL	NIL	
3	Case 3	44	M	Electrician	chennai	hypoasthesia	lower limb	nil	N	1 yr	nil	nil	Rt>Lt	Rt	Rt>Lt	Rt>Lt	S	Mn	N	N	N	N	N	N	N	grade 1	NIL	NIL	NIL	
4	Case 4	19	M	welding	thiruvallur	hypoasthesia	earlobe	nil	N swelling Neck	2months	Lt>Rt	B/L	B/L	B/L	B/L	B/L	S	Mn	N	N	N	N	N	N	N	grade 1	NIL	NIL	NIL	
5	Case 5	48	M	farmer	thruvallur	hypoasthesia	lower limb	weakness	Y	6months	B/L	B/L	B/I	B/L	B/L	B/L	SM	MMn	N	U/L	N	N	N	N	N	grade 2	NIL	Y	Y	
6	Case 6	36	M	Farmer	thiruvallur	parasthesia	lower limb	weakness	N	1 1/2yrs	nil	B/L	B/L	B/L	B/L	B/L	SM	Mn	N	N	N	N	N	N	N	grade 1	NIL	NIL	NIL	
7	Case 7	30	M	farmer	thiruvallur	hypoasthesia	both limbs	weakness	N	1 1/2yrs	nil	Rt	Rt>Lt	Rt>LT	B/L	B/L	SM	MMn	N	N	N	N	N	N	N	grade 1	NIL	NIL	NIL	
8	Case 8	21	M	literate	bihar	hypoasthesia	upper limb	weakness	N	8yrs	nil	LT>RT	B/L	B/L	B/L	B/L	SM	Mn	y	N	N	N	N	N	N	grade 1	NIL	NIL	Y	
9	Case 9	36	M	Security	kancheepuram	hypoasthesia	upper limb	nil	N	8months	nil	LT>RT	B/L	Lt>Rt	B/L	nil	S	Mn	N	N	N	N	N	N	N	grade 1	NIL	NIL	Occ	
10	Case 10	5	Fch	child	thiruvallur	nil	lower limb	nil	Y	4months	nil	nil	nil	B/L	Lt>rt	Lt>Rt	S	Mn	N	N	N	N	N	Y	N	grade 2	NIL	NIL	NIL	
11	Case 11	5	Fch	child	tanjavore	nil	lower limb	nil	Y	4mnths	nil	nil	B/L	B/L	B/L	Rt>Lt	S	Mn	N	N	N	N	N	Y	N	grade 2	NIL	NIL	NIL	
12	Case 12	45	F	housewife	thiruvallur	nil	lower limb	nil	Y	3yrs	nil	LT>RT	B/L	B/L	B/L	B/I	S	Mn	N	N	N	N	N	Y	N	grade 2	NIL	NIL	NIL	
13	Case 13	35	M	lorry driver	chennai	hypoasthesia	both limbs	nil	N	10yrs	nil	B/L	B/L	B/L	B/L	rt side	S	MMn	N	N	N	N	N	N	N	grade 1	NIL	Occ	NIL	
14	Case 14	44	M	coollie	chennai	parasthesia	lower limb	weakness	Y	5yrs	nil	B/L	B/L	B/L	B/L	rt side	SM	MMn	N	U/L	N	N	N	N	N	grade 2	5yrs,OHA	NIL	Y	**Dapsone mono,1yr 20yrs back**
15	Case 15	30	M	clerical worker i	chennai	hypoasthesia	upper limb	nil	N	6months	B/L	Lt	nil	Lt>Rt	nil	nil	S	Mn	N	N	N	N	N	N	N	grade 1	NIL	NIL	NIL	
16	Case 16	50	M	pappad fry	thiruvallur	hypoasthesia	lower limb	nil	N	2mnths	nil	Lt>Rt	B/L	B/L	B/L	B/L	S	Mn	N	N	N	N	N	N	N	grade 1	NIL	Y	Y	
17	Case 17	21	F	student	chennai	hypoasthesia	upper limb	weakness	N	3mnths	B/L	B/L	B/L	B/L	B/L	B/L	SM	Mn	N	N	N	N	N	N	N	grade 1	NIL	NIL	NIL	
18	Case 18	30	F	unemployed	thiruvallur	hypoasthesia	both limbs	weakness	Y	15yrs	B/L	B/L	B/L	B/L	Lt>Rt	Lt>Rt	SM	MMn	Y	B/L	N	N	N	Y	Y	grade 2	NIL	NIL	NIL	
19	Case 19	27	M	hotel supervisor	chennai	hypoasthesia	upper limb	nil	Y	2yrs	nil	Lt>Rt	B/L	Lt>Rt	B/L	Lt>Rt	S	Mn	Y	N	N	N	N	Y	Y	grade 2	NIL	NIL	NIL	
20	Case 20	26	M	carpenter	kancheepuram	hypoasthesia	upper limb	weakness	Y	6mths	B/L	B/L	B/L	B/L, ndlr, t	B/L	nil	SM	Mn	N	N	N	N	N	Y	N	grade 2	NIL	NIL	NIL	
21	Case 21	60	F	housewife	chennai	nil	both limbs	weakness	Y	2 ½ yrs	nil	nil	B/L	nil	B/L	B/L	SM	MMn	Y	N	B/L	N	N	N	N	grade 2	NIL	NIL	NIL	
22	Case 22	63	M	farmer	vellore	hypoasthesia	lower limb	weakness	Y	1yr	nil	Lt>Rt	B/L	B/L	B/L	B/L	SM	Mn	N	N	U/L	N	N	N	N	grade 2	NIL	Occ	Occ	
23	Case 23	21	M	weaver	thiruvallur	hypoasthesia	upper limb	weakness	Y	3yrs	B/L	Rt>Lt	B/L	Rt>Lt	B/L	B/L	SM	Mn	Y	U/L	N	N	N	N	N	grade 2	NIL	NIL	NIL	MDT-PB,RFT, 2yrs back
24	Case 24	24	F	student	sivakasi	hypoasthesia	upper limb	weakness	Y	1yr	nil	Lt>rt	nil	Lt>Rt	nil	nil	SM	Mn	Y	U/L	N	N	N	N	N	grade 2	NIL	NIL	NIL	
25	Case 25	50	F	teacher	chennai	hypoasthesia	upper limb	weakness	N	2yrs	B/L	B/L	Lt>rt	Rt>Lt	nil	Lt>Rt	SM	Mn	Y	N	N	N	N	N	N	grade 1	3yrs,OHA	NIL	NIL	MDT-PB,RFT,2yrs back
26	Case 26	64	F	housewife	chennai	hypoasthesia	lower limb	weakness	Y	9mnths	nil	nil	B/L	nil	B/L	B/L	S	Mn	Y	N	U/L	N	N	N	N	grade 2	NIL	NIL	NIL	
27	Case 27	50	M	biryani shop	chennai	nil	upper limb	weakness	Y	1yr	nil	B/L	B/L	B/L	B/L	B/L	Mo	Mn	N	U/L	N	N	N	N	N	grade 2	8yrs, OHA	NIL	Occ	
28	Case 28	29	M	metal worker	chennai	hypoasthesia	lower limb	nil	pedal edema	1yr	nil	nil	Lt>rt	Lt>Rt	Lt>Rt	Lt>Rt	S	Mn	N	N	N	N	N	N	N	grade 1	NIL	NIL	NIL	
29	Case 29	44	M	garage	bihar	parasthesia	upper limb	weakness	N	20 days	B/L	Rt>Lt	nil	Rt>Lt	nil	nil	S	Mn	N	N	N	N	N	N	N	grade 1	NIL	Y	NIL	
30	Case 30	34	F	housewife	chennai	hypoasthesia	upper limb	weakness	Y	6mnths	nil	Rt>Lt	nil	Rt>Lt	B/L	nil	SM	Mn	N	B/L	N	N	N	N	N	grade 2	2mnths,	NIL	NIL	
31	Case 31	40	F	housewife	chennai	nil	lower limb	nil	Y	1 ½ yrs	nil	nil	nil	B/L	B/L	B/L	S	Mn	N	N	N	N	N	N	N	grade 1	NIL	NIL	NIL	MDT-PB, 2006 for 3mnths irregular
32	Case 32	30	F	housewife	chennai	hypoasthesia	lower limb	nil	N	15 days	B/L	B/L	nil	Lt>Rt	Lt>Rt	B/L	S	Mn	N	N	N	N	N	N	N	grade 1	NIL	NIL	NIL	
33	Case 33	41	M	farmer	chennai	hypoasthesia	upper limb	weakness	N	1yr	nil	Lt>Rt	nil	Rt>Lt	nil	Lt>Rt	S	Mn	N	N	N	N	N	N	N	grade 1	NIL	NIL	NIL	MDT-PB,1mnth 1.5 yrs back
34	Case 34	45	M	Shop keeper	vellore	hypoasthesia	upper limb	nil	N	6mnths	nil	B/L	B/L	Lt>Rt,	B/L	B/L	S	Mn	N	N	N	N	N	N	N	grade 1	NIL	Occ	Occ	
35	Case 35	46	M	clerk	dindugal	hypoasthesia	upper limb	weakness	N	2yrs	nil	B/L	B/L	B/L	B/L	B/L	SM	Mn	Y	N	N	N	N	N	N	grade 1	4yrs, OHA	NIL	NIL	
36	Case 36	34	M	construction	thiruvallur	hypoasthesia	lower limb	nil	Y	1yr	nil	B/L	B/L	Lt>Rt	B/L	B/L	S	Mn	N	N	N	N	N	Y	N	grade 2	NIL	NIL	NIL	
37	Case 37	50	M	construction	chennai	hypoasthesia	lower limb	weakness	Y	1yr	B/L	B/L	B/L	B/L	B/L	B/L	S	Mn	Y	N	N	N	N	Y	N	grade 2	NIL	Y	Occ	
38	Case 38	25	M	electric binding	patna	parasthesia	lower limb	nil	traumatic fissure	2yrs	nil	Rt>Lt	Rt>Lt	Rt>Lt	Rt>Lt	B/L	S	Mn	N	N	N	N	N	N	N	grade 1	NIL	NIL	NIL	
39	Case 39	29	M	plumber	thiruvallur	hypoasthesia	lower limb	nil	N	6mnths	B/L	nil	B/L	B/L	B/L	B/L	S	Mn	N	N	N	N	N	N	N	grade 1	NIL	Occ	NIL	
40	Case 40	30	M	driver	thiruvallur	hypoasthesia	both limbs	weakness	Y	6mnths	nil	B/L	Rt>Lt	Lt>Rt	B/L	B/L	SM	MMn	N	B/L	B/L	B/L	N	N	N	grade 2	NIL	NIL	Y	
41	Case 41	22	F	housewife	chennai	parasthesia	upper limb	weakness	N	2mnths	nil	Lt>Rt	nil	Lt>Rt	nil	nil	S	Mn	N	N	N	N	N	N	N	grade 1	NIL	NIL	NIL	
42	Case 42	25	M	weaver	kancheepuram	nil	upper limb	weakness(pure motor)	N	3mnths	nil	Rt>Lt	nil	Rt>Lt	nil	B/L	Mo	Mn	N	N	N	N	N	N	N	grade 1	NIL	NIL	NIL	
43	Case 43	34	M	construction	kancheepuram	hypoasthesia	both limbs	nil	N	6mnths	LT>Rt	B/L	nil	B/L	B/L	B/L	S	MMn	N	N	N	N	N	N	N	grade 1	NIL	Y	NIL	
44	Case 44	30	M	cook	cudallore	hypoasthesia	upper limb	nil	N	10yrs	nil	B/L	nil	B/L	nil	nil	S	Mn	N	N	N	N	N	N	N	grade 1	NIL	Occ	Y	
45	Case 45	52	M	weaver	vellore	nil	lower limb	nil	Y	3mnths	lt side	B/L	B/L	B/L	B/L	B/L	S	Mn	N	N	N	N	N	Y	N	grade 2	NIL	Occ	Occ	
46	Case 46	48	F	housewife	chennai	nil	upper limb	weakness	Y	6yrs	nil	B/L	B/L	B/L	B/L	B/L	SM	Mn	Y	U/L	N	N	N	N	N	grade 2	5yrs,OHA	NIL	NIL	
47	Case 47	29	F	housewife	thiruvallur	hypoasthesia	upper limb	nil	N	6months	lt side	nil	B/L	B/L	B/L	B/L	S	Mn	N	N	N	N	N	N	N	grade 1	NIL	NIL	NIL	
48	Case 48	47	M	farmer	villipuram	nil	lower limb	nil	Y	12 days	B/L	nil	B/L	B/L	B/L	nil	S	Mn	N	N	N	N	N	Y	N	grade 2	NIL	NIL	NIL	
49	Case 49	21	M	construction	orissa	nil	lower limb	nil	U/L leg swelling	6yrs	B/L	nil	B/L	Rt>Lt	B/L	B/L	S	Mn	N	N	N	N	N	N	N	grade 1	NIL	Occ	NIL	
50	Case 50	45	M	farmer	vellore	hyperesthesia	both limbs	nil	N	8mnths	nil	B/L	B/L	B/L	B/L	B/L	S	MMn	N	N	N	N	N	N	N	grade 1	NIL	NIL	Y	

sl.no	Cases	Age (yrs)	Sex	Occupation	Address	Presenting Complaints					taneous Nerves involve			erve Trunks Involv			type of nerve involvement		Motor	Deformities							Disability	comorbidity			Treatment
						Sensory	extremity involved	Motor	Deformities	duration	GAN	RCN	SN	UN	LPN	PTN	N. Funct	Clinical Pattern	wasting	CH	FD	WD	FP/L	Tr.Ulcer	Resorptn	(WHO 1988)	DM	Alcohol	smoking	
51	Case 51	55	M	Porcelein manufactr	thiruvallur	nil	both limbs	wkness	Y	8yrs	nil	Lt>rt	nil	B/L	B/L	Lt	SM	MMn	wasting	B/L	nil	nil	nil	nil	nil	grade 2	nil	nil	Occ	nil
52	Case 52	29	M	pasteur church	thiruvallur	hypoesthesia	both limbs	nil	N	8yrs	nil	B/L	B/L	B/L	B/L	B/L	S	MMn	absent	nil	nil	nil	nil	nil	nil	grade 1	nil	nil	nil	Dapsone monotherapy 7yrs back
53	Case 53	35	M	metal worker	thiruvallur	Parasthesia	both limbs	wkness	Y	10yrs	B/L	B/L	B/L	B/L	B/L	B/L	SM	Po	wasting	B/L	U/L	nil	nil	present	present	grade 2	nil	Y	Y	nil
54	Case 54	48	M	clerk	vellore	Parasthesia	upper limb	normal	N	10days	nil	B/L	nil	B/L	B/L	B/L	S	Mn	absent	nil	nil	nil	nil	nil	nil	grade 1	nil	nil	Occ	nil
55	Case 55	45	M	coolie	villipuram	hypoesthesia	lower limb	wkness	claw Toes	1yr	nil	nil	B/L	B/L	B/L	B/L	SM	MMn	wasting	nil	nil	nil	nil	present	nil	grade 2	nil	nil	nil	nil
56	Case 56	26	M	machinerry	thiruvallur	hypoesthesia	lower limb	wkness	Y	15yrs	nil	nil	nil	B/L	B/L	nil	SM	Mn	wasting	nil	U/L	nil	nil	nil	nil	grade 2	nil	nil	nil	nil
57	Case 57	35	F	housewife	vellore	hypoesthesia	lower limb	nil	N	3yrs	nil	B/L	B/L	B/L	B/L	B/L	S	Mn	absent	nil	nil	nil	nil	nil	nil	grade 1	nil	nil	nil	nil
58	Case 58	50	M	farmer	kancheepuram	Parasthesia	upper limb	wkness	Y	10days	nil	nil	nil	Rt, T	nil	nil	SM	Mn	absent	U/L	nil	nil	nil	nil	nil	grade 2	nil	nil	Y	nil
59	Case 59	39	M	electrical wiring	chennai	hypoesthesia	upper limb	nil	Y	3days	B/L	B/L	B/L	B/L	nil	B/L	SM	Mn	absent	U/L	nil	nil	nil	nil	nil	grade 2	nil	Y	nil	nil
60	Case 60	27	M	railways	thiruvallur	hypoesthesia	both limbs	wkness	Y	1yr	nil	B/L	B/L	B/L	nil	B/L	SM	MMn	absent	U/L	nil	nil	nil	nil	nil	grade 2	nil	Y	nil	nil
61	Case 61	50	M	security	chennai	hypoesthesia	lower limb	wkness	Y	6yrs	B/L	nil	B/L	B/L	B/L	Lt	SM	Mn	wasting	nil	U/L	nil	nil	nil	nil	grade 2	nil	Y	Y	nil
62	Case 62	52	M	hotel worker	chennai	hypoesthesia	lower limb	Wkness	Y	5yrs	B/L	nil	B/L	B/L	B/L	B/L	SM	Mn	wasting	nil	B/L	nil	nil	nil	nil	grade 2	nil	Y	Y	nil
63	Case 63	55	M	security	thiruvanamalai	Parasthesia	lower limb	wkness	Y	1 month	nil	B/L	nil	B/L	B/L	nil	SM	Mn	absent	nil	U/L	nil	nil	nil	nil	grade 2	nil	nil	nil	nil
64	Case 64	24	M	farmer	thiruvallur	nil	upper limb	wkness	N	6mnths	U/L It	B/L	nil	B/L	nil	nil	SM	Mn	absent	nil	nil	nil	nil	nil	nil	grade 1	nil	nil	nil	nil
65	Case 65	38	F	housewife	chennai	hyperesthesia	upper limb	nil	N	1yr	nil	B/L	nil	B/L	nil	nil	S	Mn	absent	nil	nil	nil	nil	nil	nil	grade 1	nil	nil	nil	MDT-PB,RFT,1.5yrs back
66	Case 66	48	M	auto driver	thiruvallur	nil	upper limb	wkness	Y	4yrs	nil	B/L	nil	B/L	B/L	nil	Mo	Mn	wasting	U/L	nil	nil	nil	nil	nil	grade 2	nil	Y	Y	nil
67	Case 67	36	M	construction	villipuram	hypoesthesia	both limbs	nil	N	10mnths	nil	B/L	B/L	B/L	B/L	B/L	S	Mn	absent	nil	nil	nil	nil	nil	nil	grade 1	nil	nil	Y	nil
68	Case 68	35	M	carpenter	vellore	nil	both limbs	wkness	Y	15yrs	nil	B/L	B/L	B/L	B/L	B/L	SM	MMn	wasting	B/L	nil	nil	nil	nil	nil	grade 2	nil	Occ	Occ	nil
69	Case 69	38	F	construction	vellore	nil	lower limb	wkness	Y	3mnths	nil	B/L	nil	B/L	B/L	B/L	SM	Mn	absent	nil	U/L	nil	nil	nil	nil	grade 2	nil	nil	nil	nil
70	Case 70	47	M	coolie	thiruvallur	hypoesthesia	lower limb	nil	Y	1yr	nil	nil	nil	B/L	B/L	B/L	S	Mn	absent	nil	nil	nil	nil	present	nil	grade 2	nil	Occ	Occ	nil
71	Case 71	23	M	Plumber	kancheepuram	hypoesthesia	upperlimb	nil	N	1yr	nil	B/L	B/L	B/L	nil	nil	S	Mn	absent	nil	nil	nil	nil	nil	nil	grade 1	nil	Occ	nil	nil
72	Case 72	38	M	farmer	chennai	hypoesthesia	upper limb	nil	Y	6mth	nil	B/L	B/L	B/L	B/L	B/L	S	Mn	absent	nil	nil	nil	nil	present	nil	grade 2	nil	Occ	Occ	nil
73	Case 73	30	F	coolie	thiruvallur	hypoesthesia	upper limb	nil	N	1month	nil	Rt>LT	Lt>rt	B/L	B/L	nil	S	Mn	absent	nil	nil	nil	nil	nil	nil	grade 1	nil	nil	nil	nil
74	Case 74	30	M	cement worker	chennai	hypoesthesia	both limbs	nil	Y	6mnths	B/L	Rt	Rt	Rt	B/L	B/L	S	MMn	absent	nil	nil	nil	nil	present	nil	grade 2	nil	Occ	Occ	nil
75	Case 75	50	M	clerk	chennai	Parasthesia	upper limb	wknesss	N	6mnths	nil	nil	nil	B/L	nil	nil	SM	Mn	wasting	nil	nil	nil	nil	nil	nil	grade 1	nil	nil	Y	nil
76	Case 76	50	M	cement worker	chennai	hypoesthesia	lower limb	nil	Y	1yr	Lt	B/L	B/L	B/L	B/L	It	S	Mn	absent	nil	nil	nil	nil	present	nil	grade 2	nil	Y	Y	nil
77	Case 77	60	F	housewife	thiruvallur	hypoesthesia	lower limb	nil	N	2yrs	nil	nil	Lt>rt	nil	B/L	B/L	S	Mn	absent	nil	nil	nil	nil	nil	nil	grade 1	nil	nil	nil	nil
78	Case 78	39	M	farmer	thiruvallur	hyperesthesia	upper limb	nil	N	2wks	nil	nil	nil	Rt>lt	Lt>rt	nil	S	Mn	absent	nil	nil	nil	nil	nil	nil	grade 1	nil	Occ	Occ	nil
79	Case 79	36	M	hotel worker	chennai	hyperesthesia	upper limb	nil	N	20days	nil	Rt>LT	nil	Rt>lt	B/L	nil	S	Mn	absent	nil	nil	nil	nil	nil	nil	grade 1	nil	nil	nil	nil
80	Case 80	23	M	clerk-project sector	thiruvallur	nil	neck	nil	N	1month	B/L	Rt	Rt	B/L	B/L	B/L	nil	N enlargement	absent	nil	nil	nil	nil	nil	nil	grade 0	nil	nil	nil	nil
81	Case 81	45	M	auto driver	chennai	nil	upper limb	wkness	Y	15yrs	B/L	B/L	Lt>rt	B/L	B/L	Lt>Rt	SM	Mn	wasting	B/L	nil	nil	nil	present	present	grade 2	nil	Occ	Occ	nil
82	Case 82	29	M	welding	thiruvallur	hypoesthesia	both limbs	nil	Y	2yrs	B/L	B/L	B/L	B/L	B/L	B/L	S	MMn	absent	nil	nil	nil	nil	present	nil	grade 2	nil	nil	nil	nil
83	Case 83	17	F	housewife	kancheepuram	hypoesthesia	upperlimb	wkness	Autoamputn	2yrs	nil	B/L	Lt>rt	B/L	B/L	nil	SM	Mn	wasting	U/L	nil	U/L	nil	present	present	grade 2	nil	nil	nil	nil
84	Case 84	35	F	housewife	andhra	hypoesthesia	upperlimb	nil	Spontn blistrng	2yrs	nil	B/L	B/L	B/L	B/L	B/L	S	MMn	absent	nil	nil	nil	nil	present	nil	Grade 2	nil	nil	nil	nil
85	Case 85	22	M	construction	thiruvallur	hyperesthesia	lower limb	nil	N	2yrs	nil	nil	B/L	nil	nil	nil	S	Mn	absent	nil	nil	nil	n	nil	nil	grade 1	nil	Occ	Occ	nil
86	Case 86	24	M	Plumber	bihar	nil	upper limb	wkness	N	1yr	Lt	B/L	B/L	B/L	B/L	B/L	Mo	Mn	wasting	nil	nil	nil	nil	nil	nil	grade 1	nil	nil	nil	PB 6mths back, RFT,1.5yrs back
87	Case 87	35	F	housewife	thirvanamalai	hypoesthesia	both limbs	wkness	Y	1yr	B/L	B/L	B/L	B/L	B/L	B/L	SM	Po	wasting	nil	B/L	nil	nil	nil	nil	grade 2	nil	nil	nil	nil
88	Case 88	53	M	security	vellore	hyperesthesia	both limbs	wkness	Y	2yrs	nil	B/L	B/L	B/L	B/L	nil	SM	MMn	wasting	nil	B/L	nil	nil	present	present	grade 2	DM-5yrs OHA	nil	nil	nil
89	Case 89	42	M	farmer	chennai	hypoesthesia	lower limb	nil	Y	10yrs	B/L	nil	B/L	B/L	B/L	B/L	S	Po	absent	nil	nil	nil	nil	present	nil	grade 2	nil	nil	nil	nil
90	Case 90	28	F	coolie	thiruvallur	hypoesthesia	lower limb	nil	Y	6yrs	B/L	B/L	Rt>Lt	B/L	B/L	B/L	S	Po	absent	nil	nil	nil	nil	present	nil	grade 2	nil	Occ	Occ	nil

sl.no	Cases	SSS-AFB	NCS	Nerve Tissue Damage			Cellular Infiltration			Pattern of Infiltration			Necrosis	PVI	AFB	Type	SKIN
				N F L/ D	Fi	PT	E.C	G.C	F.C	Gr	D/G.I	F/S.I					
1	Case 1	Nil	SMAN	Y	N	N	N	N	Y	N	N	Y	N	N	Y	LL	
2	Case 2	Nil	SAN	N	N	N	N	N	N	N	N	Y	N	N	N	TT	
3	Case 3	Nil	SAN	N	N	N	Y	N	N	Y	N	N	N	N	N	TT	NIL
4	Case 4	Nil	SAN	N	N	Y	N	N	Y	N	N	N	N	N	N	LL	
5	Case 5	Nil	SAN	N	N	N	N	N	Y	N	N	Y	N	N	N	LL	NS
6	Case 6	Nil	SAN	N	N	Y	Y	N	N	Y	N	N	N	N	N	TT	
7	Case 7	Nil	NIL	N	N	Y	N	N	N	N	N	Y	N	N	N	TT	NIL
8	Case 8	Nil	SAN	N	Y	Y	N	N	N	N	N	N	N	N	N	TT	
9	Case 9	Nil	SAN	Y	N	N	N	N	N	N	N	N	N	Y	N	NS	
10	Case 10	Nil	NIL	N	N	N	N	N	N	N	N	Y	N	N	N	TT	
11	Case 11	Nil	SMAN	N	N	N	N	N	N	N	N	Y	N	N	N	NS	
12	Case 12	Nil	MN	N	N	N	Y	N	N	N	N	Y	N	N	N	TT	
13	Case 13	Nil	SAN	Y	N	N	N	N	N	N	N	N	N	N	N	NS	
14	Case 14	Nil	SMAN	Y	Y	N	N	N	N	N	N	Y	N	N	N	BT	
15	Case 15	Nil	NIL	Y	N	N	N	N	N	N	N	Y	N	N	N	TT	NIL
16	Case 16	Nil	NIL	N	Y	Y	N	N	N	N	N	N	Y	Y	N	TT	
17	Case 17	Nil	NIL	Y	N	N	N	N	N	N	N	N	N	N	N	NS	NIL
18	Case 18	Nil	SMAN	Y	N	Y	N	N	N	N	N	Y	N	N	N	BT	
19	Case 19	Nil	SMAN	N	N	Y	N	N	Y	N	N	N	N	N	N	BL	NS
20	Case 20	Nil	SAN	N	N	N	Y	N	N	Y	N	N	N	N	N	BT	
21	Case 21	Nil	SMAN	Y	Y	N	N	N	N	N	N	N	N	N	N	NS	
22	Case 22	Nil	SMAN	N	N	N	N	N	N	N	N	Y	N	N	N	TT	
23	Case 23	Nil	MN	N	N	N	N	N	N	N	N	Y	N	N	N	TT	NIL
24	Case 24	Nil	SAN	N	N	N	N	N	N	N	Y	N	N	N	N	BT	
25	Case 25	Nil	SMAN	N	N	N	Y	N	N	Y	N	N	N	N	N	TT	
26	Case 26	Nil	SAN	N	N	N	N	N	N	N	N	N	N	N	N	NIL	NIL
27	Case 27	Nil	SMAN	Y	N	N	N	N	Y	N	N	N	N	Y	N	BT	
28	Case 28	Nil	MN	N	N	N	Y	N	N	N	N	Y	N	Y	N	TT	
29	Case 29	Nil	SMAN	N	N	N	Y	N	N	N	Y	N	N	Y	N	TT	
30	Case 30	Nil	SMAN	N	N	N	N	N	N	N	Y	N	N	N	Y	BT	NIL
31	Case 31	Nil	SMAN	N	N	N	Y	N	N	N	N	Y	N	N	N	TT	
32	Case 32	Nil	NIL	N	Y	N	N	N	N	Y	N	N	N	N	N	TT	
33	Case 33	Nil	SMAN	N	N	N	N	N	N	N	N	N	N	N	N	NIL	NIL
34	Case 34	Nil	SMAN	N	N	N	N	N	N	N	N	N	N	N	N	NIL	
35	Case 35	Nil	SMAN	Y	Y	Y	N	N	N	N	N	N	N	N	N	TT	
36	Case 36	Nil	MN	N	N	N	N	N	N	N	Y	N	N	N	N	BT	NS
37	Case 37	Nil	MN	N	Y	N	N	N	N	N	N	N	N	N	N	NS	
38	Case 38	Nil	NIL	N	N	N	N	N	N	N	N	Y	N	Y	N	BT	
39	Case 39	Nil	NIL	N	N	Y	N	N	N	N	Y	N	N	Y	N	TT	
40	Case 40	Nil	SAN	N	N	Y	Y	N	Y	N	Y	N	N	N	N	BT	NIL
41	Case 41	Nil	MN	N	N	Y	N	N	Y	N	N	Y	N	N	N	TT	
42	Case 42	Nil	MN	Y	N	N	N	N	N	N	N	N	N	N	N	NS	
43	Case 43	Nil	SMAN	N	N	N	Y	N	N	N	N	N	N	N	N	TT	
44	Case 44	Nil	NIL	Y	N	Y	N	N	N	N	N	N	N	Y	N	TT	
45	Case 45	Nil	SAN	N	N	N	Y	N	N	N	N	Y	N	N	N	TT	
46	Case 46	Nil	SMAN	Y	Y	N	N	N	N	N	N	N	N	N	N	TT	
47	Case 47	Nil	NIL	N	N	N	N	N	N	N	N	Y	N	N	N	TT	NIL
48	Case 48	Nil	NIL	N	N	N	N	N	N	N	Y	N	N	Y	N	BT	NIL
49	Case 49	Nil	NIL	N	N	N	Y	N	N	N	N	Y	N	N	N	TT	NIL
50	Case 50	Nil	SAN	Y	N	N	Y	N	N	N	N	Y	N	N	N	TT	

COLOUR PLATES

P1a. Pure Neuritic Leprosy- Auto-amputation with claw hand

P1b.Pure Neuritic Leprosy- Auto-amputation with claw hand, Above Patient

P2a.Greater Auricular Nerve – Cord like thickening,

P2b. Greater Auricular Nerve- Same patient

P3. Bilateral Claw Hand with Wasting of Intrinsic Muscles of Hand

P4. Bilateral Claw Hand- with both Sensory-Motor Involvement

P5. Wrist Drop with muscle wasting

P6. Foot Drop- Left Foot

P7. Bilateral Facial Nerve Palsy-Lagophthalmos

P8. Pure Neuritic Leprosy-Claw Toes

P9. Pure Neuritic Leprosy-Resorption of Left Index Finger

P10. Resorption with Claw hand and Muscle Wasting

P11. Trophic Ulcer with Resorption of index and fourth finger

P12. Trophic Ulcers over the Pressure Areas

P13a. Enlarged Cutaneous Nerves

P13b. Nodular Tender Cutaneous Nerve Enlargement

[redacted]

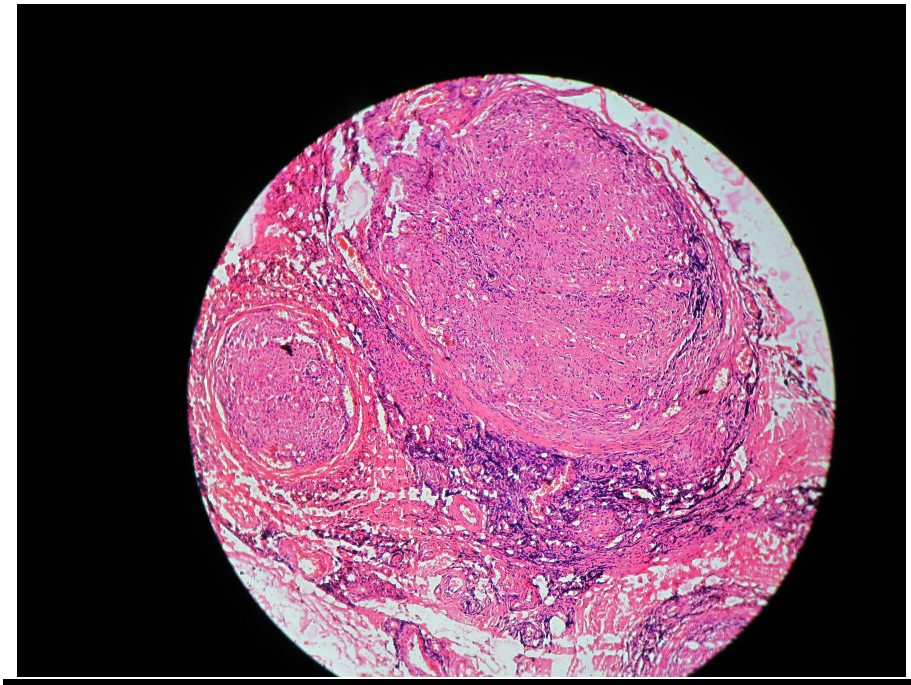
P14a. Trophic Ulcer- 5 Year old female child

P14b. Trophic Ulcer –Same Patient

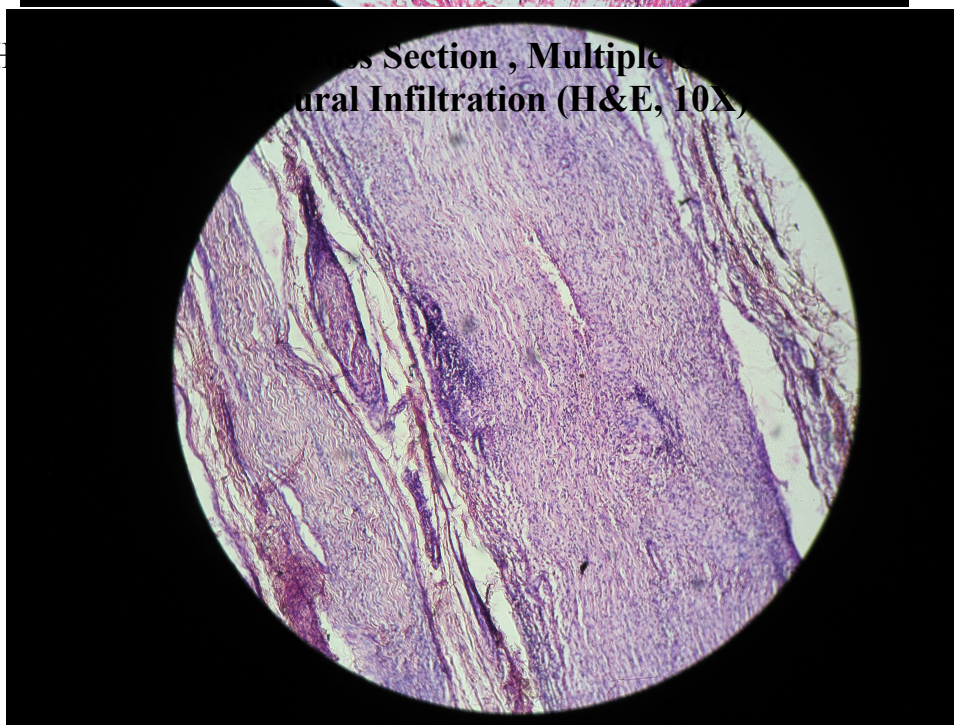
P15. Spontaneous Blistering- Ulceration

P16. Trophic Ulcer Lateral Malleolus

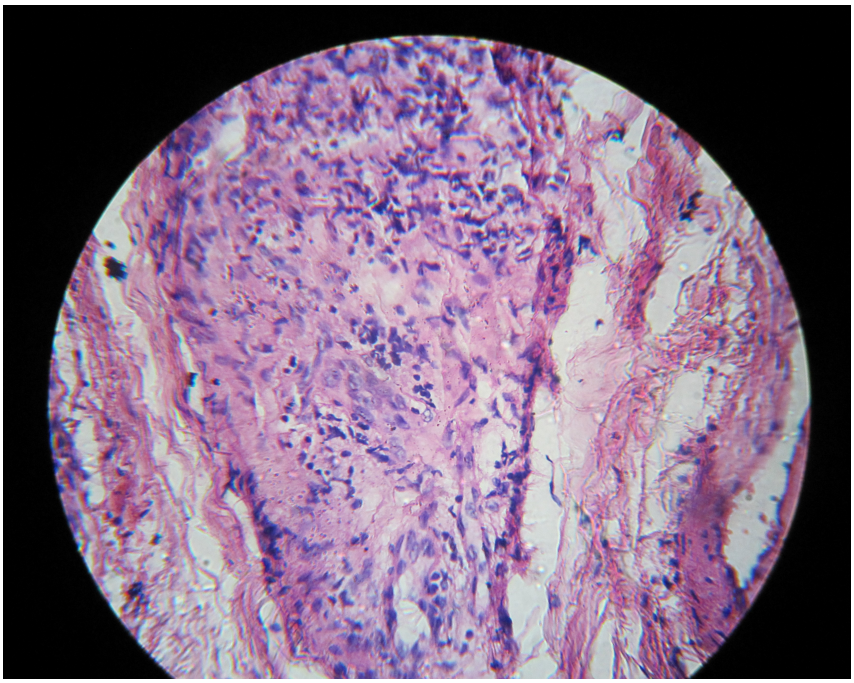
P17. Left Sural Nerve Biopsy



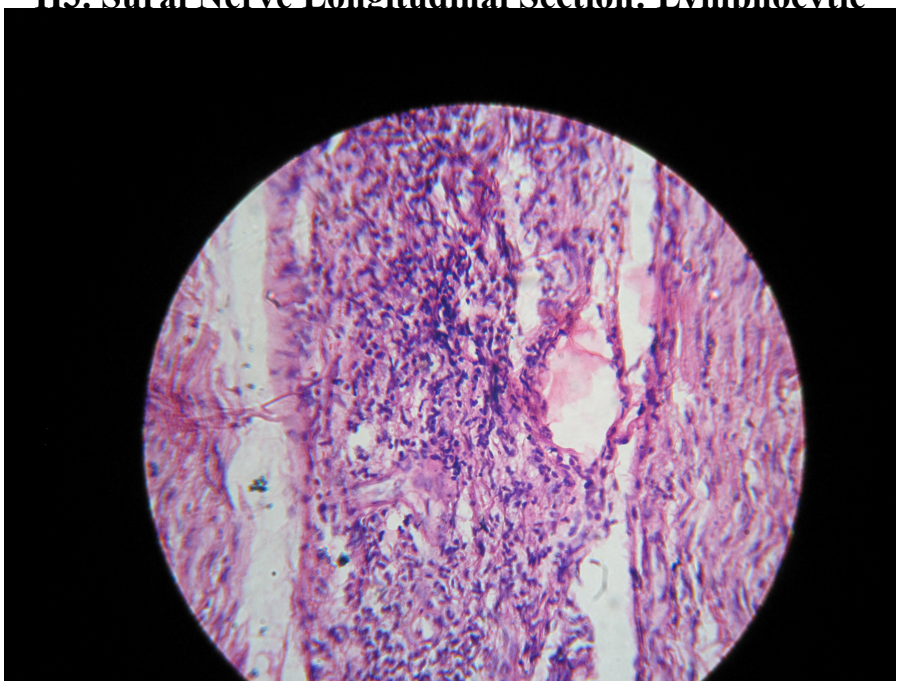
H ss Section , Multiple
ural Infiltration (H&E, 10X)



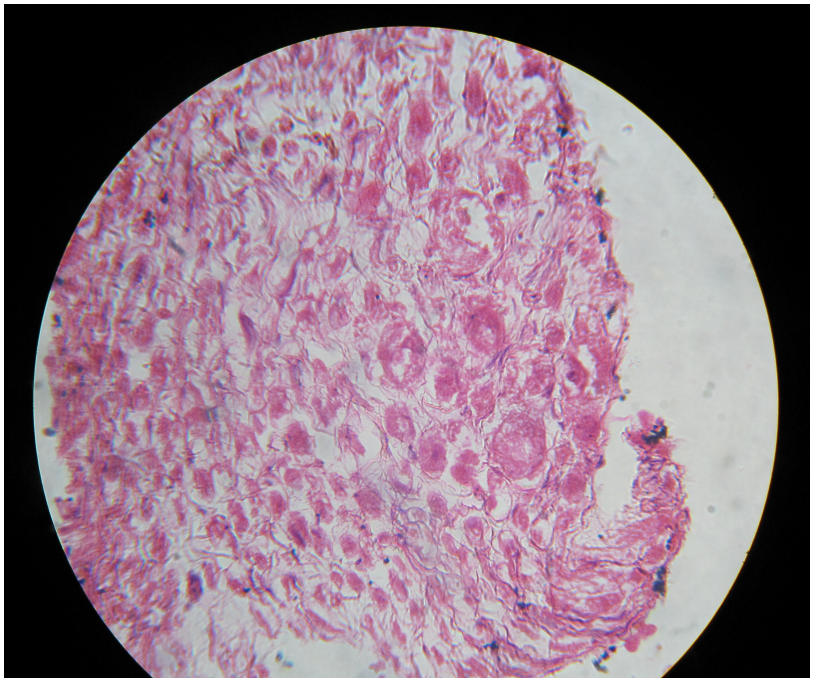
H2. Sural Nerve Longitudinal Section: Diffuse Infiltration with Few Granulomas (H&E, 10X)



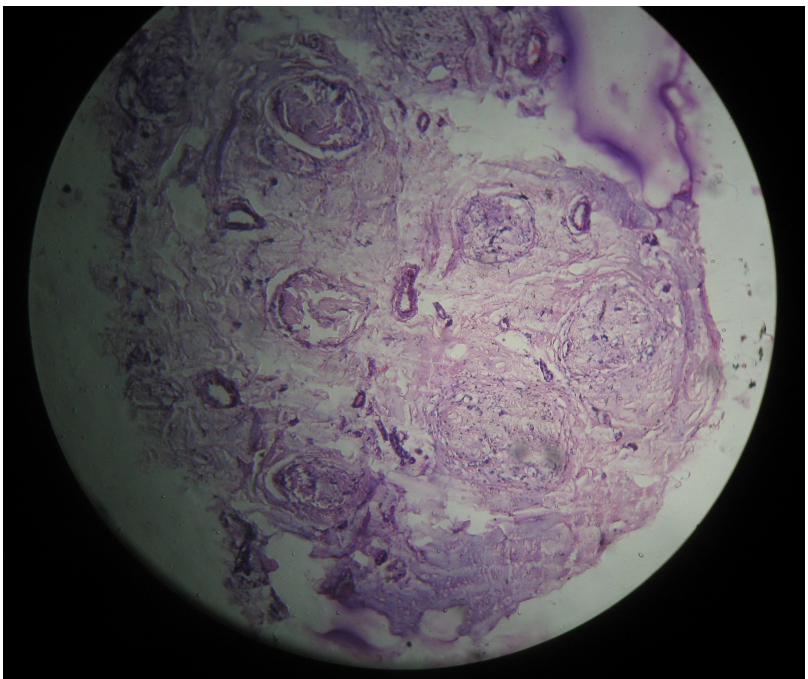
H3. Sural Nerve Longitudinal Section: Lymphocytic



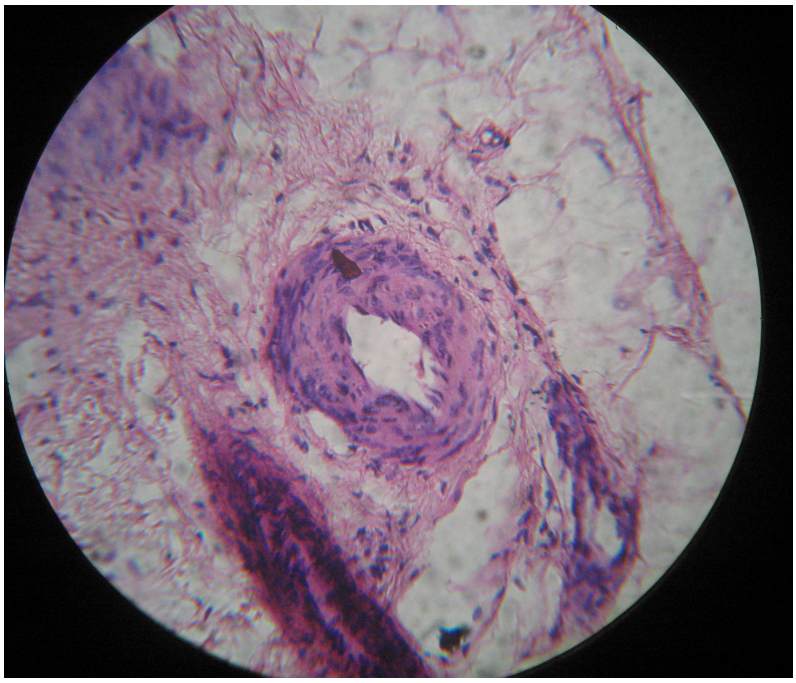
H4. Sural Nerve Longitudinal Section: Diffuse Infiltration with Giant Cells (H&E, 10X)



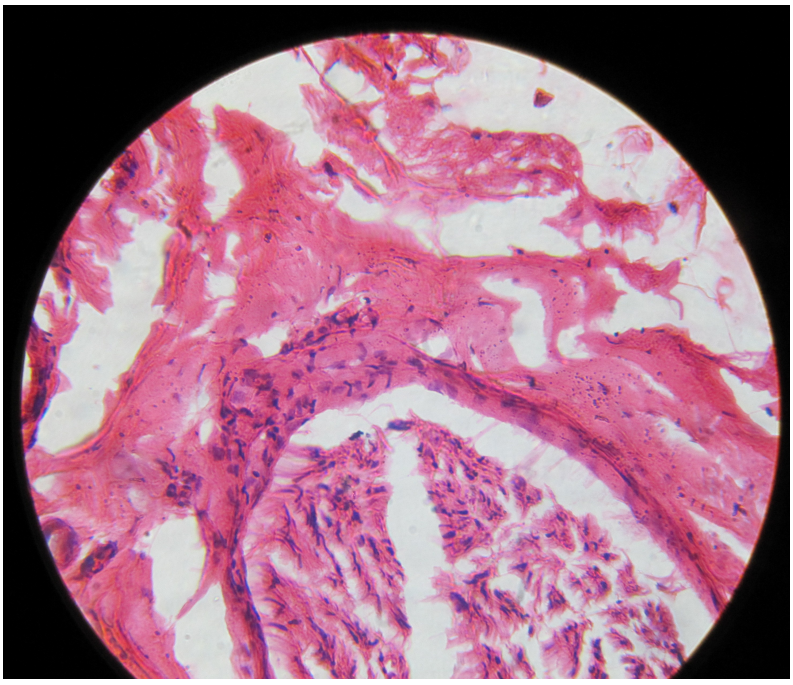
H5. Sural Nerve – Cross Section , Axonal Degneration with Nerve Fibre Loss and Perineural Thickening (H&E, 10X)



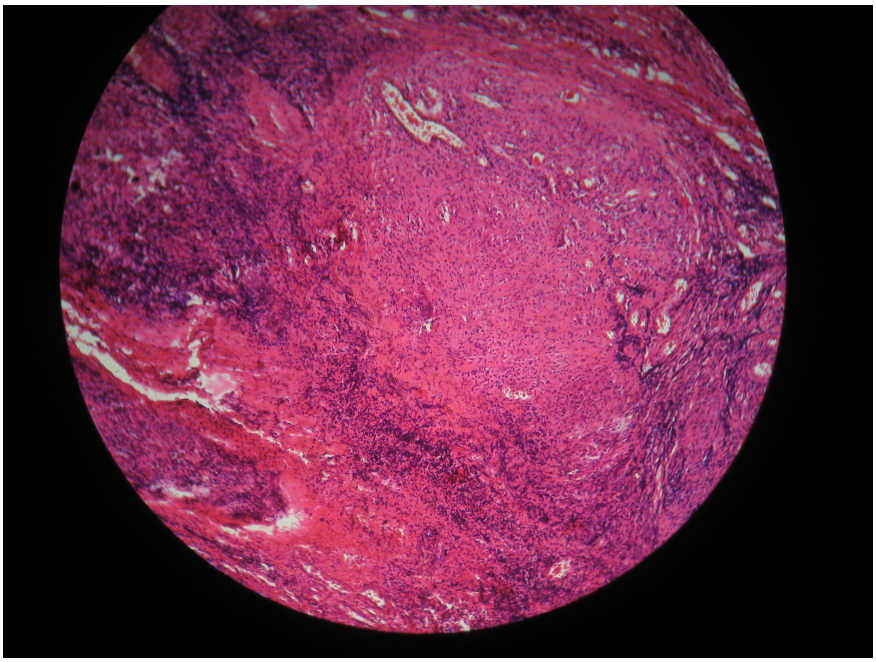
H6. Sural Nerve – Cross Section : Nerve Fibre Loss and Perineural Fibrosis (H&E, 10X)



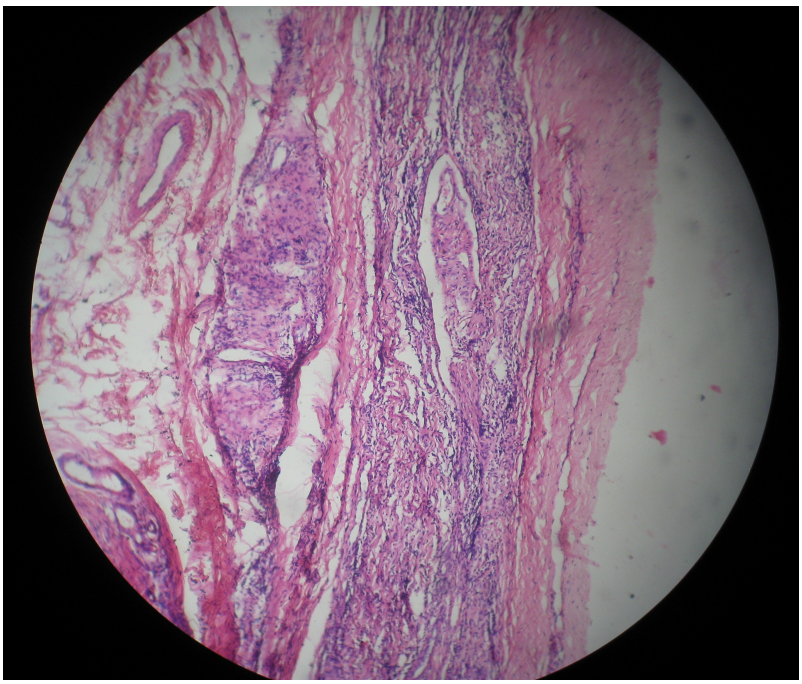
H7. Sural Nerve – Longitudinal Section : Perivascular Infiltration (H&E, 10X)



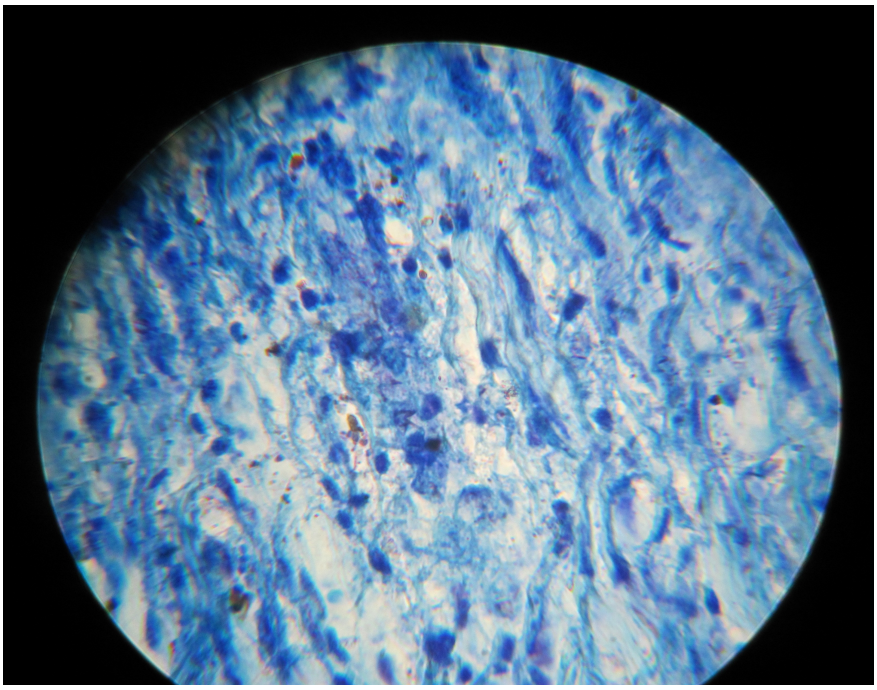
H8. Sural Nerve – Cross Section: Perineural infiltration (H&E, 40X)



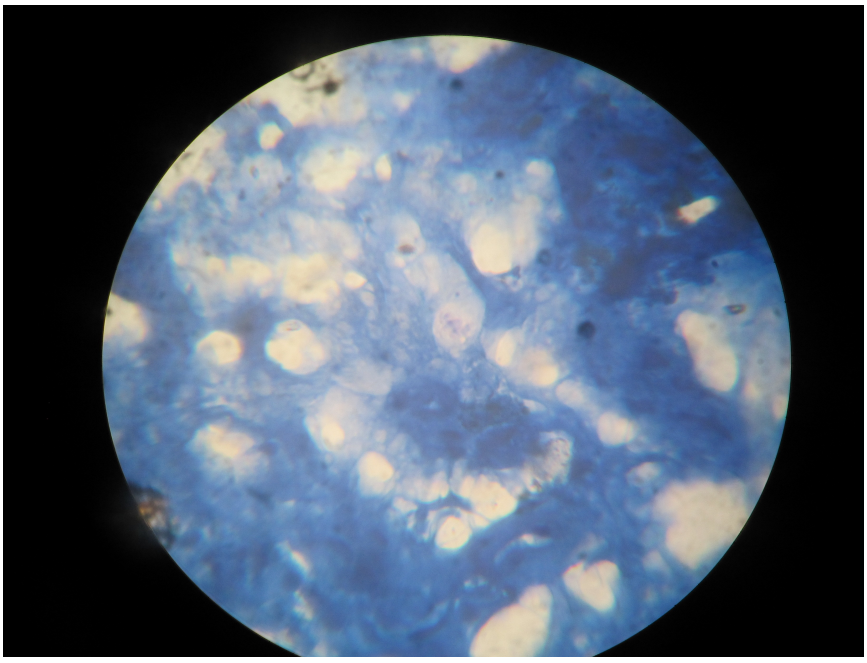
H9. Sural Nerve – Cross Section : Diffuse Iniltration, Borderline type (H&E, 10X)



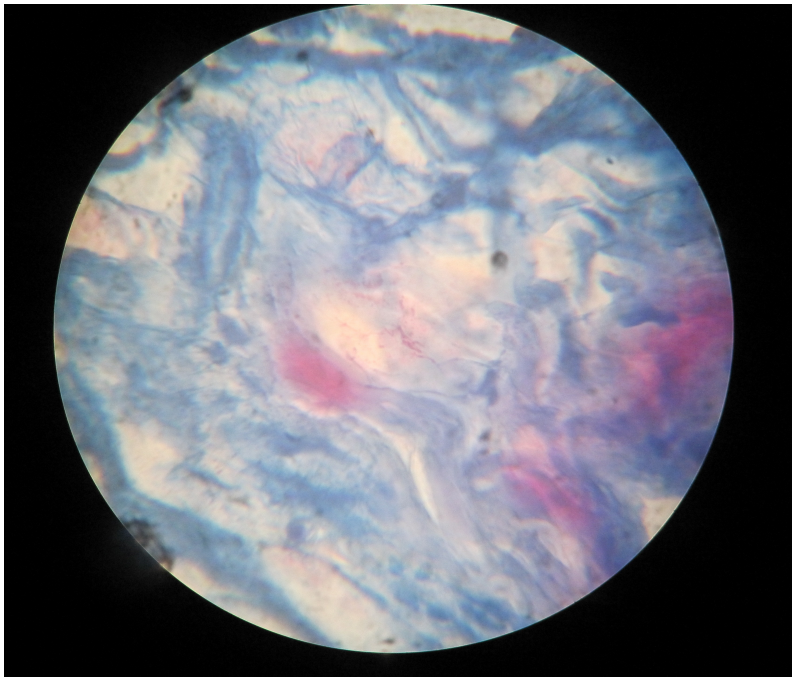
H10. Sural Nerve – Longitudinal : Diffuse infiltration, Borderline type (H&E, 10X)



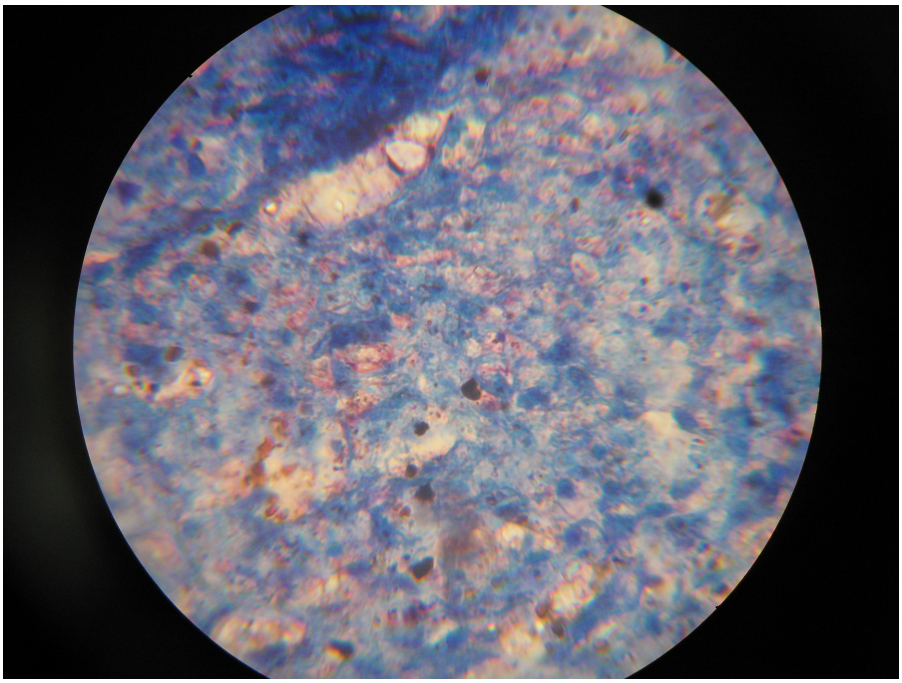
H11. Sural Nerve – Longitudinal Section : M.leprae, Borderline (F&F,oil, 100X)



H12. Sural Nerve : Foamy Macrophages, (F&F,oil, 100X)



H13. Sural Nerve : M.leprae, (F&F,oil, 100X)



H14. Sural Nerve – Longitudinal :M.leprae and Foam Cells, Lepromatous type (F&F, oil, 100X)

PROFORMA

Case no:	Age:	OP/IP no:
Name:	Sex:	H.O.P No:
Occupation:	D.O.A	D.O.D:
Address:	Religion:	Date:

Previous Treatment of Hansen: Still on treatment/Completed/Discontinued

Corticosteroid therapy: Duration

H/O Smoking/Alcohol/Exposure to STD

H/o DM/HTN/TB/Epilepsy/Immunosuppressive disease:

Nerve Symptoms **Duration**

Acute/Chronic/Recurrent

Pain/Swelling

Sensory

Motor:

Cranial

Autonomic:

Associated symptoms:

Fever:

Arthralgia:

Musculoskeletal Pain:

Edema of Hands/Feet/Face:

Pain in Palms/Soles:

Lymphadenopathy:

Rhinitis/Epistaxis/Other nasal Symptoms:

Appearance of New Lesions:

Systemic Symptoms:

Eyes Gastrointestinal

Gonadal/Urinary Others

PHYSICAL EXAMINATION:

Pulse BP

Pallor/Cyanosis/Tetanus/Clubbing/Edema/Lymphadenopathy

Temperature Eyes:

Mucosa Erythema/tenderness of palms/Soles

Dactylitis: Epidydimo Orchitis:

Bones & Joints: Tenosynovitis:

Skin Changes:

Nerve Examination:

Sensory:
Glove & Stocking anaesthesia/Fine/Crude/Vibration/Pain/Temperature

Extremities		Upper Limb	Lower Limb
	Rt:		
	Lt:		
Motor:		Upper Limb Rt Lt	Lower Limb Rt Lt
	Bulk:		
	Tone:		
	Power:		
Reflexes:		Upper Limb	Lower Limb
	Rt:	bi/tri/sup	knee/ankle/plantar
	Lt:	bi/tri/sup	knee/ankle/plantar

Palpation of Nerves		Upper Limb	Lower Limb
	Rt:	SO/IO/GA/SC/Ulnar/Radial/Median	CP/Sural/PT
	Lt:	SO/IO/GA/SC/Ulnar/Radial/Median	CP/Sural/PT

(+=Palpable,t=Tender)

Cranial Nerves:

Nail: Hair:

Deformities:

Paralytic Deformities:

Claw hand	Bilateral/Unilateral
Wrist Drop	Bilateral/Unilateral
Foot Drop	Bilateral/Unilateral
Claw toes	Bilateral/Unilateral
Facial Nerve Palsy/Lagophthalmos	Bilateral/Unilateral

Anesthetic Deformities:

Trophic Ulcers

Spontaneous Blistering

Auto Amputation

Resorption

Investigations:

CBC:	ELISA
RBS:	Chest Xray
LFT:	VDRL:
RFT:	
SSS-AFB	
Nerve Biopsy: H& E	Fite Faraco
Skin Biopsy H& E	Fite Faraco